A.M.A.

# Archives OF OTOLARYNGOLOGY

Significance of [illegible] in Persistent Respiratory Infection
*Arthur Dintenfass*

Direct Aden[illegible]
*Paul Guggenheim*

New Technique [illegible] of the Deviated Nasal Septum
*Irving B. Goldman*

Scleroma
[illegible]*nburg and F. C. MacCarthy*

Tinnitus, Deaf[illegible]igo
*Edmund Prince Fowler*

Surgery of Ca[illegible] Larynx
*Henry Boylan Orton*

Associated La[illegible]
*Antonio Rullan*

An Experimen[illegible] Audiometry in Simulated Partial Dea[illegible]
*Lawrence K. Gundrum*

Continuous, or [illegible]ency, Audiometry
*H. A. E[illegible]son Dishoeck*

Fenestra Oval[illegible]ic Deafness
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SEPTEMBER 1956
VOLUME 64 NUMBER 3

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# TABLE OF CONTENTS

VOLUME 64 SEPTEMBER 1956 NUMBER 3

## ORIGINAL ARTICLES

Significance of Allergy in Persistent Respiratory Infection — PAGE
*Arthur Dintenfass, M.D., Atlantic City, N. J.* ..... 171

Direct Adenoidectomy
*Paul Guggenheim, M.D., Topeka, Kan.* ..... 178

New Technique in Surgery of the Deviated Nasal Septum
*Irving B. Goldman, M.D., New York* ..... 183

Scleroma
*L. W. Falkinburg, M.D., and F. C. MacCardell, M.D., Providence, R. I.* 190

Tinnitus, Deafness, and Vertigo
*Edmund Prince Fowler, M.D., New York* ..... 196

Surgery of Carcinoma of the Larynx
*Henry Boylan Orton, M.D., Newark, N. J.* ..... 205

Associated Laryngeal Paralysis
*Antonio Rullan, M.D., M.Sc. (Med.), San Juan, Puerto Rico* ..... 207

## AUDIOLOGY SECTION

An Experimental Study of Audiometry in Simulated Partial Deafness
*Lawrence K. Gundrum, M.D., Los Angeles* ..... 213

Continuous, or Sweep-Frequency, Audiometry
*H. A. E. van Dishoeck, M.D., Leyden, Netherlands* ..... 216

Fenestra Ovalis for Otosclerotic Deafness
*Samuel Rosen, M.D., New York* ..... 227

## CASE REPORTS

Rhabdomyosarcoma of Hypopharynx
*Rudolph W. Pearson, M.D.; E. B. Gammell, M.D., and Walter Thayer, M.D., Providence, R. I.* ..... 238

## CLINICAL NOTES, NEW INSTRUMENTS AND TECHNIQUES

Urea in Ménière's Disease, Migraine, and Allied Conditions
*Irving Muskat, M.D., Milwaukee* ..... 241

## REGULAR DEPARTMENTS

Abstracts from Current Literature ..... 243
News and Comment ..... 212
Books ..... 250
Directory of Otolaryngological Societies ..... 251

# A. M. A.
# ARCHIVES OF OTOLARYNGOLOGY

VOLUME 64 SEPTEMBER 1956 NUMBER 3



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Published monthly by the AMERICAN MEDICAL ASSOCIATION. Editorial and Circulation Offices: 535 North Dearborn Street, Chicago 10, Illinois. Publication Office: Thompson Lane, Box 539, Nashville 1, Tennessee. Change of Address: Notice to the circulation office should state whether or not change is permanent and should include the old address. Six weeks' notice is required to effect a change of address. Second-class mail privileges authorized at Nashville, Tenn., Aug. 6, 1956.



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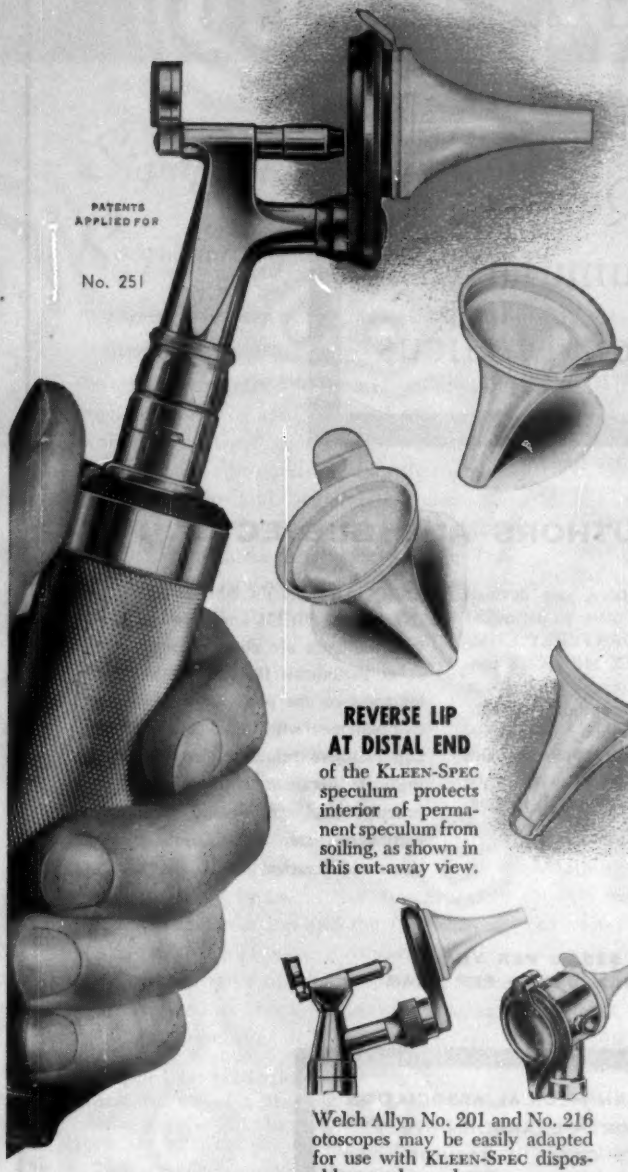


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A.M.A. Archives OF OTOLARYNGOLOGY

# Significance of Allergy in Persistent Respiratory Infection

ARTHUR DINTENFASS, M.D., Atlantic City, N. J.

Respiratory-tract allergy and pathological variations due to associated infection occur more frequently than generally recognized. It is now a well-established fact that allergy predisposes to and aggravates many diseases of the upper and lower respiratory tract. Recent surveys * relating to the frequency of allergy as an etiologic factor or as a complication in diseases of the ear, nose, and throat show 70%-80% of all office patients to be allergic. Kern [4] states that the respiratory tract is the site of the commonest, the most diversified, and some of the severest manifestations of hypersensitivity. Respiratory-tract allergy encompasses such a vast field that only the theoretical high lights and important clinical applications will be included.

The primary factor in the clinical aspect of the allergic reaction and the fundamental concept with which we are concerned is that the various parts of the respiratory tract be considered as shock organs. It is at any or all of these sites that the allergic reaction takes place after the exposure of the tissue antibody to the antigen.

Respiratory allergy rarely exists alone. Allergic rhinitis, whether perennial or seasonal in the form of hay fever, and bronchial asthma may be purely allergic in their onset or acute phase but almost immediately or coincidently are complicated by infection. In order to understand the relationship of infection to respiratory allergy, it is necessary to be cognizant of the pertinent physiology.

## Physiologic Considerations

The nose, constituting the natural airway for respiration, has three primary functions:

1. Olfaction.

2. Conditioning the inspired air to the requirements of the pulmonary surfaces by warming, moistening, and filtering.

3. Cleansing itself of the foreign material extracted from the air. In the normal nose, ciliated mucous membranes form a continuous lining from the vestibule to the nasopharynx. A sheet of tenacious mucous secretion acts as a conveyor belt, overlying the cilia, propelled by the cilia from every part of the nose and sinuses of the pharynx. Ciliary pathways in the sinuses are always toward and through the ostia; in the nose, toward the pharynx; in the bronchial tree, toward the glottis. Foreign particles (including bacteria) impinge upon the mucous sheet, which is moved posteriorly into the

Accepted for publication March 21, 1956.

Amplification of a presentation made at a staff meeting of the Atlantic City Hospital, Dec. 28, 1955.

From the Otolaryngology Department, University of Pennsylvania Graduate School of Medicine, and the Departments of Allergy and Otolaryngology, Atlantic City Hospital.

* References 1-3.

nasopharynx by the cilia, aided by gravity, traction, and the act of swallowing. Here the mucus, known as "postnasal drip," makes contact with the widely dispersed lymphoid tissue, the first line of defense of the upper respiratory tract.

Lymph tissue and lymphocytes play an important role in resistance. Lymph tissue is the probable site of the immune response and antibody formation, as evidenced by a convincing number of experiments.[5] Lymphocytes function in the attenuation of bacterial and viral agents; at the same time the nodes which they form act as a filtering bed for the removal of infective and particulate material.

Schenck [6] states that the plasma cell appears to be the only cellular element in lymphoid tissue capable of synthesizing antibodies and gamma globulin; further, that this is the prime function of the lymphoid tissues of Waldeyer's ring because of its strategic position and the lymphoid structural modification.

## Pathology

For practical purposes respiratory allergy can be considered a catarrhal inflammation characterized by tissue edema with eosinophilic infiltration and a mucoserous discharge of glandular and goblet-cell origin. The edema may be transitory and disappear with the removal of the allergenic irritant. When the allergen persists, the edema becomes pronounced and the stroma becomes overloaded. In the nose and sinuses polyps are thereby formed. In the bronchi there is edematous thickening of the mucous membrane and hypertrophy of the musculature.

Kline [7] has stated that the characteristics of infection in allergic patients are rapid onset, violent course, and slow regression. He finds the tissue changes in allergy may be considered under five headings: (1) functional changes, (2) inflammatory changes, (3) proliferative changes, (4) degenerative changes, and (5) necrotic changes.

The chronic infection so commonly found in the allergic person causes an increase in lymph tissue due to hyperplasia and infiltration of fibroblasts. As a result of the progressive hyperplasia, the lymph tissue extends far beyond the original confines of the nodule and approaches the muscle fibers below. In time, obstruction of lymph channels and lymph vessels occurs by invasion of fibroblasts, construction of fibrous tissue, and proliferation of endothelium within the lymph channels. Bacterial allergy and inhalant sensitivity likewise stimulate this lymphoid-tissue hypertrophy, which is so prevalent in respiratory allergy.

## Etiology

A great deal of factual and experimental evidence [8] has been presented to demonstrate the role of allergy in the epidemiology of the common cold. Exposure to cold, humidity, fatigue, and debility are associated with lowered resistance to the common cold, and all severely influence the allergic state. The increased frequency of other allergies in a cold-susceptible person and in the members of his family suggests a specific allergy to the virus or its protein. It is a well-recognized fact that the early symptoms and clinical findings in the common cold and allergic rhinitis are similar and frequently indistinguishable.

Recent studies † have shown that perennial allergic rhinitis is the most important underlying factor in upper respiratory disorders, such as sinusitis, pharyngitis, and postnasal discharge. Bacterial allergy, alone or in association with inhalant or extrinsic factors, is an important cause of these conditions.

Schenck [11] was among the first to describe the role of allergy in the etiology of chronic sinusitis. He finds that the ethmoid and maxillary sinuses are more prone to become involved in allergic reactions than frontal or sphenoidal sinuses because of their close relationship to the turbinates, which are considered as the primary shock organs. Grove [12] believes that sinus disease is a very

† References 3, 4, 9, and 10.

important cause of asthma. Van Alyea [13] vividly demonstrates the importance of allergy in chronic sinus infection. He states that allergic edema and polyps block the nose and diminish the size of the sinus ostia to a minimum, thereby aiding in the inception and maintenance of attacks of sinusitis.

Allergy has been shown to be of utmost importance in recurrent ear diseases.‡ The tissues of the nasopharynx, Eustachian tube, and middle and external ears may become shock areas to offending allergens. Secretory otitis media and secondary suppurative otitis media following Eustachian tubal closure with allergic edema are commonly seen. Also, allergic edema of the nasopharynx increases the patient's susceptibility to secondary infection. The combination of allergy and infection in otitis externa makes this entity one of the commonest and most difficult to treat in general otolaryngologic practice. The infectious factors must be controlled before the allergic elements will respond.

Davison [16] has pointed out the high incidence of allergy in bronchopulmonary infections, including chronic bronchitis, bronchiectasis, and allergic pulmonary consolidation, or Löffler's syndrome. He relates bacterial protein sensitization in allergic persons to the necrotic type of allergic reaction described by Kline, and feels it is this reaction that is responsible for many cases of bronchiectasis.

Bacterial allergy is considered to be intrinsic in its origin, resulting from activity of bacterial antigens directly upon the respiratory mucosa. Locally, the allergic reaction probably depends upon the formation of a bacterial hapten, the mucosa acting as a shock organ after being originally sensitized by some pathogenic organism.

Since an alteration of the allergic state is so often involved, a vicious cycle is established. The allergy lowers the resistance of the mucous membranes and interferes with ventilation and drainage, so that secondary infection becomes superimposed. This, in turn, increases the allergic tendency with bacterial factors.

‡ References 14 and 15.

### Clinical Findings

Early in allergic rhinitis the mucous membranes have the classical pale, grayish-blue appearance. With the development of local bacterial infection and sensitization, a deeper-red color is acquired. Protracted infection results in hyperplastic changes and a diminution or loss of ciliary activity, with consequent impairment in the drainage of the nose and sinuses. The continuation of the process produces in time polypoid degeneration, mulberry-type pitting of the posterior tips of the turbinates, and the formation of polyps. As sequelae, there follow chronic sinusitis, nasopharyngeal infection, and Eustachian-tube and middle-ear disease.

When the allergic reaction takes place within the bronchial tree, bronchial asthma is the result, with its typical shortness of breath, cough, and wheeze. The resulting blockage has serious consequences causing dyspnea, bronchial obstruction, and often atelectasis, which, if long continued, can lead to bronchiectasis.

Examination of the patient must be thorough and include, in addition to the routine otolaryngologic examination, inspection of the nasal chambers following shrinkage and aspiration of existing secretion; transillumination of the frontal and maxillary sinuses; nasopharyngoscopic examination; finger palpation of the nasopharynx; mirror examination of the epipharynx and hypopharynx, and evaluation of hearing. X-rays of the sinuses and postnasal area demonstrate pathology when present. Chest roentgenograms and bronchograms are to be performed in selected cases. The eosinophile content of nasal, postnasal, and bronchial secretions may be significant; but the absence of eosinophiles, even after repeated smears, does not preclude the diagnosis of allergy.

## Treatment

In the treatment of respiratory infection the primary aim is to obtain adequate drainage and ventilation and to remove the underlying etiologic agent. Conservative measures are desirable and should be first used unless more radical procedures are obviously needed. These measures include local decongestant therapy to the nose and paranasal sinuses, general supportive aids (including antibiotics and vaccine therapy), and, most important, a well-planned allergic regimen. More radical procedures involve the use of surgery or irradiation in removal of infected and hypertrophied lymphoid tissue. Conservative measures may be helpful in alleviating the patient's discomfort, at least temporarily accomplishing the necessary drainage and ventilation while the more permanent allergic measures take hold.

Treatment of the allergic state is too often neglected, and the patient is studied and treated inadequately. Allergy is frequently undiagnosed or misdiagnosed, because the patient is first seen with a complicating complaint, such as sinusitis, postnasal drip, or bronchitis. Careful history often reveals a family background of allergic diseases and, in many cases, a past history of "colic" and eczema during infancy, hives, frequent colds and respiratory infections, and repeated surgical procedures involving the tonsils and adenoids in children and the nose and paranasal sinuses in adults.

Properly performed skin tests and their intelligent interpretation are essential. However, there are many overlapping factors, such as house dust, molds, infections, and food allergens, which confuse, requiring patience and time to make a complete and accurate diagnosis.

Among the specific causes of respiratory allergy the inhaled allergens are most important. House dust, bedding materials, wool and domestic animal danders, cosmetics, and pyrethrum are the commonest (listed here in the order of their importance). However, ingested substances may also be involved, especially cereals, nuts, chocolate, sea foods, milk and eggs, and foods eaten raw. Smoke, sharp odors, and fumes of paint and lacquers may be exciting causes. Sudden changes in temperature and barometric pressure, and emotional upsets at times produce aggravation of symptoms.

Skin tests for sensitivity are an essential part of the diagnostic procedure. Their value depends upon the ability of the one who selects the substances to be tested and who interprets the test results. The common inhalants must be tested in each patient. Additional inhalants depend upon the history of exposures. It is wise to test the important pollens that occur in the patient's locality. Foods may have to be tested depending upon the type of symptoms and relationship to meals, environment, and other allergic manifestations. The patient's own house dust is one test substance that should never be omitted.[4]

In the treatment of respiratory allergy there should be complete avoidance of offending allergens. Special attention must be paid to the removal of all allergenic or potentially allergenic substances from the bedroom. Covering the pillow or mattress with an "allergen-proof" casing does not establish complete avoidance of the content. Moreover, foam rubber is now readily available as a substitute. The elimination of pets from the household, books and stuffed toys from the child's bedroom, cosmetics containing orris root, and insecticides containing pyrethrum flowers are only a few of the necessary measures. Avoidance of house dust is difficult. Complete avoidance can be obtained (and at best only temporarily) by a change in residence and in furnishings. Desensitization with the patient's own house dust is usually advisable and has proved more beneficial than stock dust. Adequate diagnosis and treatment of seasonal hay fever is especially effective in reducing perennial symptoms. Many other details in allergic management, not mentioned here, are essential in the thorough treatment of these patients.

Symptomatic treatment of the upper respiratory tract during exacerbations includes the use of mild nasal decongestants and antihistaminic drugs. Caution must be observed in the use of local therapy, as prolonged use of drugs often causes irritation of the nasal membranes and continuation of symptoms. The antihistaminic drugs have proved to be of great value in symptomatic relief, but at best they are merely palliative. They should not be given over long periods of time or when diagnostic tests are performed. Careful selection of the antihistamines will produce better results as well as reduce the undesirable side-effects,[17] such as drowsiness, vertigo, irritability, nausea, and vomiting. They should be avoided in asthma, since most increase the viscosity of bronchial secretions.

In regard to the lower respiratory tract, we are more concerned with the prophylaxis of the asthmatic attack. With proper care of the patient with allergic rhinitis and/or sinusitis, or the potential asthmatic, the allergenic precipitating factor or the infectious trigger mechanism may be averted.

There is a difference of opinion concerning the value of bacterial stock and autogenous vaccines. All of us have seen many failures and some success with this type of treatment. In my experience, in carefully selected cases, where frank purulent material may be obtained from sinus ostia or adjacent areas, the use of a spot suction has proved efficacious in providing significant culture material and, consequently, a potent vaccine. Bronchial aspirations are often the source of cultures for therapeutically effective vaccines. Autogenous vaccines sometimes fail because of the presence of different bacterial flora with successive clinical infections. Thus, vaccine obtained during one infection may not be effective during succeeding infection.

Antibiotic and chemotherapeutic agents are valuable and often necessary adjuncts in the treatment of the infectious elements that complicate respiratory allergy. In most patients the anticipated therapeutic response is not obtained until the infection is completely controlled.

In the thorough treatment of the allergic patient it is necessary to consider the relationship of systemic conditions as endocrine dysfunctions, nutritional deficiencies, gastrointestinal disturbances, and psychogenic factors. In patients with severe emotional strain and physical fatigue the adaptation syndrome and alarm reaction of Selye[18] come into play. Selye has shown that there are normal physiological and biochemical reactions of the body to shock and stress, causing depletion or excessive liberation of the hormones of the adrenal cortex with profound changes in various body structures. There has been much recent progress in our concept of the mechanisms of these hormones and their action in inhibiting allergic edema and inflammation, fibrosis, and granulation tissue. Corticotropin and the various cortisone derivatives have proved to be exceedingly helpful in the treatment of stubborn and intractable respiratory allergy and its infectious complications.

Davison § has effectively used corticosteroids in infection associated with allergy. He finds that these drugs are particularly helpful in hyperplastic sinusitis and inflammatory edema of the larynx and bronchi. He states that the decrease in capillary permeability produced by these compounds results in a decrease in the inflammatory edema consistently seen in bacterial allergy.

Surgical treatment embodies the basic principles of surgery to obtain adequate drainage and remove the etiologic agent. In the nose a deviated septum or a septal spur is indication for surgical correction if there is firm lateral contact with the turbinates, since this causes interference with breathing as well as with adequate sinus drainage. In allergic patients the operation should never be performed during a pollen season, and

§ Davison, F. W.: The Use of Cortico-Steroids in Otolaryngology, presented at the Annual Meeting of the New Jersey Society of Ophthalmology and Otolaryngology, March 18, 1956.

preferably after Oct. 15 and before April 1, lest the patient develop a new sensitivity. Nasal polyps, when large enough to obstruct, must be removed regardless of season. Thorough allergic regimen usually causes regression in polypoid degeneration and frequently disappearance of small polyps. More radical procedures, such as antral punctures and windows, and sinus surgery, are necessary only if there is severe sepsis of the sinus or if there is massive polyp formation within the sinus.

In lower respiratory tract involvement bronchoscopy is indicated as a means of diagnosis and for therapeutic benefit by aspirating the viscid exudate which the patient has been unable to expectorate. In regard to diagnosis, bronchoscopy is of value in ruling out other causes of wheezing. It gives a clear idea of the amount of bronchitis present and the degree of mucosal thickening; and, more important, it furnishes an uncontaminated specimen of endobronchial exudate for smears and cultures, vaccine preparation, and sensitivity tests. The latter, of course, are important in indicating the choice of antibiotic.

## Report of Cases

Several case reports are herewith presented illustrating some of the important general principles in the diagnosis and treatment of the respiratory allergic patient. It should be noted that all have had both surgery and antiallergic therapy but never received the two concurrently. Typically, the patient with upper respiratory allergy complained of lifelong "catarrah" and "nose trouble." The chief symptom was nasal obstruction. Other complaints were sneezing, watery or purulent nasal and postnasal discharge, frequent colds, lack of energy or easy fatigue, and poor tolerance for air-conditioning and temperature changes. All had personal and family histories of allergy, and most had repeated nasal operations.

Case 1.—A 54-year-old man had repeated polypectomies performed over a 20-year period, approximately every six to eight months, plus intermittent inadequate episodes of allergic therapy. Nasal drainage had been constant with an abundant thick yellow discharge. Examination demonstrated markedly congested pale turbinates with polypoid degeneration. Massive polyps completely obstructed both nasal chambers. On March 9, 1954, polypectomy and exenteration of the anterior ethmoid sinuses was performed. Desensitization was undertaken with autogenous vaccine and the patient's own house dust. There has been no recurrence of polyps in over two years, and patient states for the first time in his life he has been free of headaches and has adequate nasal ventilation.

Case 2.—A 48-year-old woman presented a long history of both perennial and seasonal hay fever and several polypectomies over a 10-year period. Polypectomy was performed March 3, 1952, and a complete allergic survey undertaken. Patient did well on a desensitization program consisting of her own house dust, and grass and ragweed extracts until the summer of 1954 when the corn blossomed in the fields surrounding her home. A right-sided obstructing nasal polyp was removed. The addition of corn pollen extract produced a symptom-free 1955 pollen season.

Case 3.—A 52-year-old man gave a history of eight polypectomies with no allergic care. Examination disclosed both nasal chambers completely occluded by large polyps. Polypectomy was performed Jan. 2, 1953, and patient placed on a regimen of dust and stock catarrhal vaccine. He continued under observation and therapy for a period of four months, during which time he was completely symptom-free. He was advised to continue his desensitization program at decreased intervals but lapsed treatment and was not seen until Dec. 10, 1955, when there was partial regrowth of the polyps. Polypectomy and resumption of the allergic program has kept this patient free of symptoms (last seen in late February, 1956).

Case 4.—A 10-year-old boy was first seen on Aug. 4, 1951, with complaints of complete left nasal obstruction and a mass protruding from the left nostril. There was a strong family history of allergy as well as a personal history of hives and seasonal ragweed hay fever. In addition, household pets included a family of cats. Skin tests demonstrated markedly positive reaction to cats in addition to house dust, feathers, and ragweed. With all due precautions, the large polyp was removed Aug. 21, 1951. Nasopharyngeal fibroma was ruled out by microscopic study. The cats were removed and allergic therapy given with dust and ragweed extracts for three months. Patient has been seen on several occasions since that time with no evidence of recurrence and minimal allergic symptoms.

Case 5.—A 55-year-old woman presented a history of recurrent intractable bronchial asthma following three severe attacks of bronchopneu-

monia during the years 1952 to 1954. Competent allergic care had been rendered without definite improvement. Bronchoscopy was performed June 1, 1955, and left bronchostenosis found approximately 5 cm. distal to the carina. This was dilated and a specimen collected for preparation of an autogenous vaccine. Patient had been entirely free of symptoms following this procedure and the reinstitution of a thorough allergic regimen, including dust and autogenous vaccine therapy.

These case reports demonstrate the successful results obtained by the combination of essentially minor surgical procedures and comprehensive allergic therapy. It is well to reemphasize that most patients respond to conservative local measures in conjunction with an allergic regimen.

## Summary

The constant association of allergy and infection affects all parts of the respiratory tract, producing diverse but characteristic patterns in the nose, paranasal sinuses, Eustachian tubes, each of the anatomical divisions of the ear, and the bronchial tree.

The underlying physiology and pathology is described, relating the importance of infection upon the immunologic and pathologic changes.

Efficient treatment is dependent upon adequate study of the allergic state. The offending allergens are strictly avoided or used in a carefully planned desensitization program.

Properly selected and timed surgical procedures are essential in affording adequate ventilation and drainage.

The use of conservative surgical procedures in conjunction with allergic therapy is stressed as being productive of maximum improvement and more permanent relief.

Representative case reports illustrative of the essential principles are briefly described.

In conclusion, an attempt has been made to demonstrate the close association and interdependence of respiratory-tract infection and allergy, and the need to have a thorough understanding of both allergic and surgical principles in order to obtain maximum therapeutic benefits.

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# Direct Adenoidectomy

## I. A Historicoliterary Review

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Direct adenoidectomy is now universally accepted among practitioners of our specialty, although armamentarium and techniques vary considerably about the country. The literature on the subject is of very modest proportions as perhaps befits so apparently commonplace a procedure. Yet, though we marvel at the advances made in the more spectacular fields of fenestration and stapedial mobilization, it is perhaps well to reflect that far more people become hearing cripples from otitis media and its sequelae than from otosclerosis. On this account, we have been wont to chide pediatricians and general practitioners for not performing more myringotomies. The workmanship of our own adenoidectomies is undoubtedly of equal importance. Consequently, the literature of adenoidectomy, though not voluminous, is of the highest significance.

Review of this literature reveals considerable lack of agreement as to the historical origins of the direct approach to adenoidectomy. Concise resumés have been given by Senturia * and myself.[3] It seemed worth while to review the history of the procedure in more detail at this time.

A. von Gyergyai, a Hungarian living in Vienna, was the first to have attempted direct visualization and surgery of the nasopharynx. In 1910 he presented[4] a method of direct access by means of a set of cylindrical tubes of various lengths and diameters, illuminated by a Bruening electroscope. With these he was able to perform operations not only upon the adenoid but also upon the sphenoid sinuses and pituitary body.

Yankauer,[5] a year later, presented his new speculum as an improvement over Gyergyai's tubes. In a classic paper he described with amazing clarity the novel features of the nasopharynx as seen under direct vision, features which many operators even today have great difficulty conceptualizing. He gave an optical explanation of the contrast between the direct and the familiar mirror view. (The reader is referred to the original paper or to my 1952 publication[3] for details.)

Edgar Holmes[6] that same year (1911) presented to the world his electric nasopharyngoscope.

Joseph Beck[7] in 1913 introduced the use of small rubber catheters, placed through the nose and hooked around the free edge of the soft palate, to obtain a limited direct view of the nasopharynx.

O. A. Lothrop, who will be mentioned further hereafter, invented a soft palate retractor in 1914.

These four men represent what might well be called the "classical period" in the history of American work upon the nasopharynx. After them comes a long dearth of contributions. L. K. Guggenheim[8] spoke of this period of neglect of the nasopharynx as "one of the darkest chapters in otolaryngology."

A virtual renaissance was ushered in by Andrew Love's[9] description of his soft palate retractor, the first to appear in the literature. This instrument, in the opinion of the author and many others, is still superior to the many retractors which are available today. Love's paper, as epochmaking as it was brief, introduced an en-

Accepted for publication May 16, 1956.

* References 1 and 2.

tirely new concept of nasopharyngeal visualization. In order to appreciate this fact it must be understood that the specula of Gyergyai and Yankauer were endoscopic instruments. Neither afforded a direct open exposure.

It is instructive in this regard to compare the problem of visualization of the nasopharynx with that of the larynx. Unlike the tubular larynx, the nasopharynx is an irregular, noncylindrical cavity which lends itself, but poorly, to an endoscopic approach. In laryngoscopy an open approach is also possible. The well-known Lynch suspension apparatus provides for actual retraction of the hypopharynx. This type of laryngoscopy is, of course, optional, whereas, in the nasopharynx, there is no real choice between endoscopy and open retraction. Unfortunately, the retraction of the soft palate cannot be accomplished by any sort of gallows. The position of the retractor requires constant dynamic adjustment. The whole procedure of exposure is, in fact, far less standardized than suspension laryngoscopy and, for that very reason, correspondingly more difficult to learn.

It is interesting, from the point of view of phylogenesis of surgical instruments, that the earliest model of the Love retractor † consisted of a hemicylindroid which was essentially a Yankauer speculum with the side of the tube opposite the handle removed. Love ingeniously employed this hemi-Yankauer as a palate retractor by holding the handle cephalad instead of caudad. The radius of curvature of the cross section was later increased so that a gentle arc evolved which was ideally adapted to the natural curvature of the nasopharyngeal surface of the soft palate. A lip was added which hooked behind the free edge of the palate, making the instrument virtually slip-proof. Love considered the angle of 110 degrees between retracting blade and handle ideal.

A remarkably similar instrument was invented by O. A. Lothrop, of Boston, in 1914, according to Meltzer,[10] who was his student. This instrument, which Meltzer still prefers, has a retracting blade practically identical in design with the Love except for the fact that it joins the handle at an angle of 90 degrees instead of 110. Also, the Lothrop retractor is made in only one size as compared with three for the Love.

Meltzer ‡ states that he recalls no publication by Lothrop of a description of his excellent instrument, and Senturia [1] and I have each had the frustrating experience of searching the literature for one in vain. There can be no doubt that Love's invention was entirely independent of Lothrop's, and the similarities of the two instruments are to be accounted for by the fact that both men must have had a very fine instinct for taking cues from nature in arriving at a unique solution to a most complex problem in surgical engineering.§ Whether Lothrop evolved his instrument from the Yankauer, as did Love, will probably remain forever an unsolved problem in the embryology of our modern armamentarium. Certainly the publication of Yankauer's [5] classic paper three years before Lothrop's invention would make this an intriguing probability.

One might be permitted a speculation on the reason for Lothrop's mysterious literary silence, a silence which can hardly be explained by the fact that his fame was already secure as a result of numerous other contributions to our specialty. Might he not modestly have hesitated because of a feeling that anyone proposing improvements in so "elementary" an operation as adenoidectomy would be committing an indiscretion and perhaps inviting ridicule? It is clear that he had a tremendous respect for the procedure which was fully a generation in advance of the body of the specialty.

Love's contribution received compara-

† Love, A. A.: Personal communication to the author.

‡ Meltzer, P. E.: Personal communication to the author.

§ Love developed his retractor in 1932 while a resident in otolaryngology at the Los Angeles County General Hospital on the service of Dr. John MacKenzie Brown.

tively little attention until L. K. Guggenheim || evolved his method of adenoidectomy based upon it. Guggenheim at first called this procedure "The G-Vault Operation," later coining the more modest term "direct adenoidectomy." He was the first to report results in children with conduction hearing impairments, many of whom had already undergone tonsillectomy-adenoidectomy. These results were eminently satisfactory, normal hearing being restored in the vast majority of cases.

A notable feature of Guggenheim's work was his description of a new clinicoanatomic entity, the lateral lymphoid curtains of the nasopharynx. These structures were shown to be quite separate from the central adenoid mass and to lie in intimate contact with the Eustachian orifices. Guggenheim described an elegant method of ablating them by sharp dissection with specially devised scissors.

Von Gyergyai published another paper,[12] in 1939 (merely cited in Senturia's ¶ bibliography), which confined itself to the problem of proper design of tubular specula for work in the nasopharynx.# No mention was made of any of the work done in America—work for which Gyergyai himself was the inspiration.

The work of Love and Guggenheim was shortly followed by a widespread revival of interest in the nasopharynx and its problems. This interest took the form of a national enthusiasm for the Crowe radium applicator.

The outstanding 1949 paper by Meltzer [10] redirected attention to surgical management and acquainted the specialty at large for the first time with the pioneer work of Lothrop. Within a couple years the potential dangers of the radium applicator came to be widely appreciated, and enthusiasm for this treatment modality declined rapidly.

Reeves and Brill [13] in a 1951 publication described in great detail the salpingopalatine, salpingonasal, and salpingopharyngeal folds and discussed the pathologic significance of hypertrophy of these structures. Meltzer * and Senturia † similarly employed the term "salpingopharyngeal folds" in referring to what L. K. Guggenheim had called "the lateral lymphoid curtains of the nasopharynx." An impression that these folds consist not of lymphoid tissue but rather of the muscular and tendinous fibers of the corresponding muscles was confirmed by reference to Morris' "Human Anatomy,"[16] which states: "A fold of mucosa descending from the posterior lip of the aperture [of the Eustachian tube] to the lateral pharyngeal wall is the *plica salpingopharyngea* due to the *musculus salpingopharyngeus.* An inconspicuous *plica salpingopalatina* descends from the anterior lip to the soft palate." No mention was made of a plica salpingonasalis.

The lateral lymphoid curtain, which alone is of surgical interest here, covers all these structures in a somewhat indiscriminate manner, although it is evidently associated most intimately with the salpingopharyngeal fold. From a purely anatomic viewpoint it would seem questionable to speak of hypertrophy of the folds themselves, since this usage would appear to imply an overgrowth of the muscular fibers of which they consist, whereas the only hypertrophy which occurs is that of the lymphoid curtain. The underlying musculotendinous folds stressed by Reeves and Brill are themselves, in fact, exceedingly inconspicuous in the surgically clean nasopharynx as seen under direct

|| References 8 and 11.

¶ References 1 and 2.

# It is interesting to note that Gyergyai has unroofed his original tubular specula posteriorly at the distal, or palatal, end and anteriorly at the proximal end, so that a hemicylindrical palate retractor was in effect joined to a second hemicylinder pressing caudally against the tongue, lower teeth, and lower lip. The specula—in four or five sizes—were designed to extend from lip to posterior nasal spine and to be fixed, if desired, to the patient's head (i. e., to be self-retaining). It is fascinating to note that Senturia's speculum has, since 1952, recapitulated the same line of development from closed cylinder (Yankauer) to fenestrated (hemi-) cylinder, now closely approaching the design of the Love.

* References 10, 14, and 15.

† References 1 and 2.

vision; and their detailed dimensional measurements given by these authors [17] could well be measurements of more or less adventitious variations in size and configuration of the overlying lymphoid curtain.

Reeves and Brill list three indications for removal of what we shall henceforth call the lateral lymphoid curtains, i. e., for "complete adenoidectomy." The first of these, which provided the subtitle to their 1951 paper, is "to overcome interference with nasal breathing by the salpingonasal and salpingopalatine folds." The concept of interference with nasal respiration by the lateral lymphoid curtains is not one shared by other surgeons. Indeed, Meltzer [14] writes: "The chief purpose of adenoidectomy is to alleviate nasal obstruction. And here it must be admitted that, when this is the only purpose, the operation even in the least skilled hands can be successful. The technique may be deplorable, yet the results be amazingly good."

Reeves and Brill's second indication is the existence of a chronic focus of infection in these structures—a matter which has received insufficient attention by others. The lateral curtains are not infrequently conspicuous in both children and adults who have had otherwise adequate tonsillectomy and adenoidectomy, and may show hypertrophy and chronic infection. Even when recurring otitis is not a problem, these infected curtains may be responsible for chronic or recurring acute inflammations of the pharynx and paranasal sinuses.

The third listed but by all odds most important indication for removal of the curtains is interference with ventilation of the tympanic cavities, predisposing to purulent otitis and progressive hearing impairment. The otologic indications have been fully discussed by L. K. Guggenheim ‡ and by Meltzer.§

My own 1952 article [3] collected in a single surgical publication historical and technical details which previously had been scattered in other contexts.

Senturia || in two thoughtful 1953 publications has contributed some sound basic thinking on the adenoid problem generally. He has evolved a palate retractor which, he believes, offers certain advantages over the Love.

Recent (1955) contributions by Manning [18] and Laszlo [19] are also concerned with efforts to design a more satisfactory palate retractor. The very multiplicity of instruments available for this purpose would appear to indicate that no one of them is entirely satisfactory. On the other hand, it seems more probable that the difficulties encountered by various operators are not basically related to inadequacies of armamentarium but rather to the problem of conceptualization of the direct view of the nasopharynx and effective manipulative technique derived therefrom.

A new impetus to better surgical management of the adenoid problem comes from the recent observation by Clark [20] of a strong association of thyroid cancer in children and adolescents with earlier irradiation for hypertrophied thymus glands or adenoids. Hitherto there was no reason to suspect that x-radiation was unsafe for the management of adenoid recurrences.

Adequate adenoid surgery is demanding of both the time and the best efforts of the otolaryngologist. Review of the available literature reveals a wide variety of techniques for which exclusive claims are made. These techniques have evolved as a result of long-continued efforts by their originators to solve one of the most difficult and generally neglected problems in our specialty. As such, they merit our profound respect. It would not seem amiss, however, to discuss these technical procedures in a constructively critical way. This will be the task of a subsequent communication.

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§ References 10, 14, and 15.

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# New Technique in Surgery of the Deviated Nasal Septum

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Deviations of the nasal septum which cause dysfunction in nasal respiration require surgical correction. The degree of the deviation and the site and structural consistency of the deflected part have, in the past, influenced the success or failure of the operation.

The development of septal surgery has passed through many phases. Ingals [1] was the first to suggest the accomplishment of a submucous operation by removal of a part of the triangular cartilage. Krieg [2] improved the technique by resecting the entire deflected cartilage. Boenninghaus [3] completed the procedure by removing the osseous septum (perpendicular plate of the ethmoid and vomer) as well as the cartilage. Surgical refinements were added by Freer,[4] Killian,[5] Hajek,[6] Ballenger,[7] Yankauer,[8] and St. Clair Thomson.[9] Submucous resection thus became the standard procedure for the rhinologist.

There were still many inadequacies in the technique. A satisfactory buttress had to be left superiorly and anteriorly to prevent saddling and retraction of the columella. The limitations of this operation have been demonstrated by the various steps for improvement suggested by different rhinologic surgeons. These ranged from simple shifting of the septum, its incomplete removal and implantation of anterior struts, to complete removal and/or replacement by cartilaginous implants. These modifications have been proposed by Metzenbaum,[10] Peer,[11] Salinger,[12] Cohen,[13] Fomon,[14] Becker,[15] Cottle,[16] Lierle and Huffman,[17] and others.

The procedure described in this presentation was devised because it was believed that it had the following advantages:

(*a*) Restoration of septal function.

(*b*) Prevention of saddling deformity.

(*c*) Prevention of nasal-tip drooping.

(*d*) Prevention of flaccidity of the septum by retaining semirigid structures anteriorly and rigid or bony structures posteriorly.

(*e*) Prevention of retraction by retaining, or regaining, a straight septum, membranous septum, and columella.

(*f*) Lessened vulnerability to subsequent trauma.

(*g*) Obviated need of free grafts for replacement, camouflage, or support.

The objective in the operation is the restoration of normal anatomic relationship of the deflected septum without, or minimal, sacrifice of its structures. The essential principle in the operation consists in mobilization of the septum in the midline by use of the following:

(*a*) Pedicle flaps of deflected caudal sections.

(*b*) Central replacement of posterior sections.

(*c*) Resection, if necessary, of a small cartilaginous or bony bulge which may be obstructive.

## Technical Steps

The operation should be accomplished with the patient supine and the head slightly

Accepted for publication May 3, 1956.

From the Division of Rhinoplasty, Otolaryngological Service, Mount Sinai Hospital.

Read before the Section on Laryngology, Otology, and Rhinology at the 104th Annual Meeting of the American Medical Association, Atlantic City, June 7, 1955.

elevated. Barbiturates are administered preoperatively. Adults are operated upon under local anesthesia with 1.5% butethamine (Monocaine) and epinephrine, 1:30,000, unless there is drug sensitivity or other contraindications.

For a successful result it is necessary to proceed as follows:

1. A low right unilateral columellar incision is made at the caudal edge of the septum and intercolumellar tissue is separated.

2. A mattress suture is inserted at the columellar base (Fig. 1*A*, *B*, *C*, and *D*).

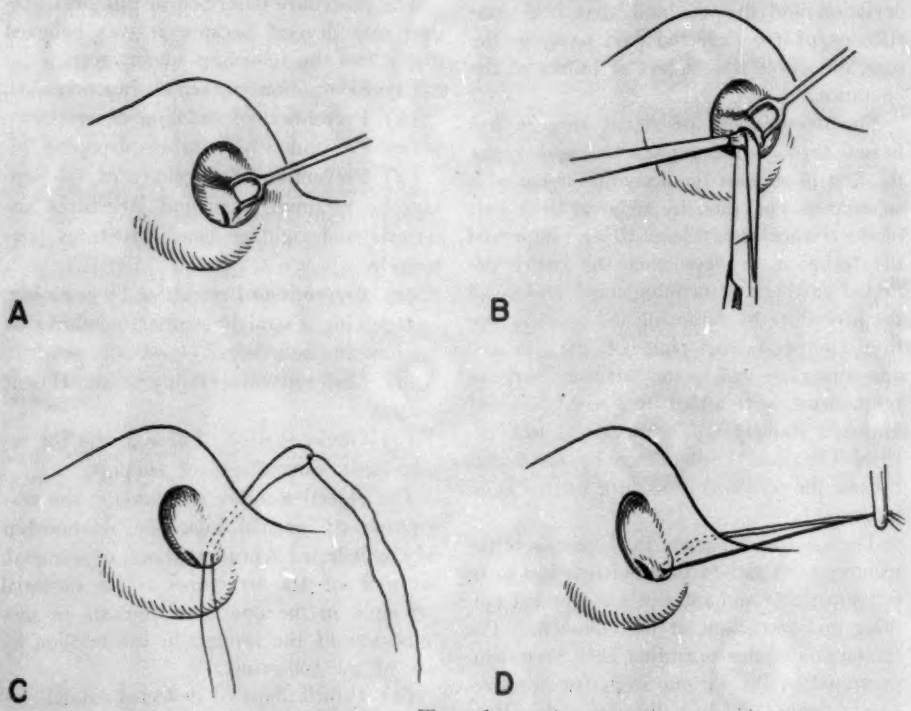


Figure 1

3. The septum is completely transfixed.

4. The septum is bilaterally freed by elevating the mucoperichondrium through the transfixion incision (Fig. 2*A*, *B*, *C*, *D*, and *E*).

5. The deviated caudal component is converted into two vertical sections.

6. The entire septum is displaced to the midline, posterior to the formed sections, by fracturing the osseous part (Fig. 3*A*, *B*, *C*, and *D*).

7. Slight resection, if necessary, of overlapping or angulating cartilaginous septum, ridge, or thickened vomer.

8. Anterior nasal spine is replaced in midline; if too wide, it is completely or partly removed (Fig. 4 *A* and *B*).

9. Aligned vertical caudal sections are reattached to mucoperichondrium.

10. Septum is sutured to columella (Fig. 5*A*, *B*, *C*, and *D*).

11. Nose is packed with petrolatum gauze.

## Comment

Submucous resection became the usual procedure for the deviated septum at the turn of this century. Since then the evolution of the operation has passed through three major phases that were based upon new anatomic and physiologic concepts and mechanical principles. The leading pro-

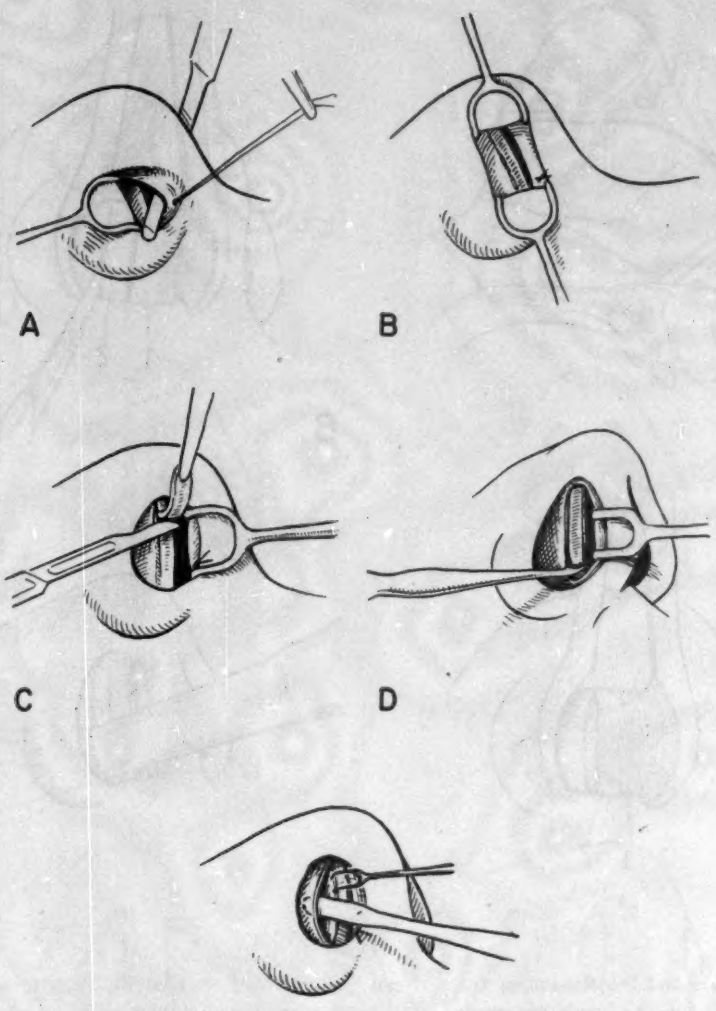


Figure 2

ponents of each modification in techniques were (*a*) Freer or Killian, (*b*) Metzenbaum, and (*c*) Fomon.

The Killian operation was unsatisfactory because the ventral part of the cartilage was left for supportive purposes and to maintain the nasal profile. The deflection of the luxated triangular cartilage, where the anterior edge appears in the vestibule, could not be corrected, although obstructive, because of the support it gave to the tip and columella.

Metzenbaum's procedure was the first advance in reconstructive surgery of the caudal deflection. It was a "swinging-door" variety of operation in which the cartilage incised at the angle of deflection produced a mobile segment for replacement in the columella. The operation was limited in scope because the mucoperichondrium was liberated only

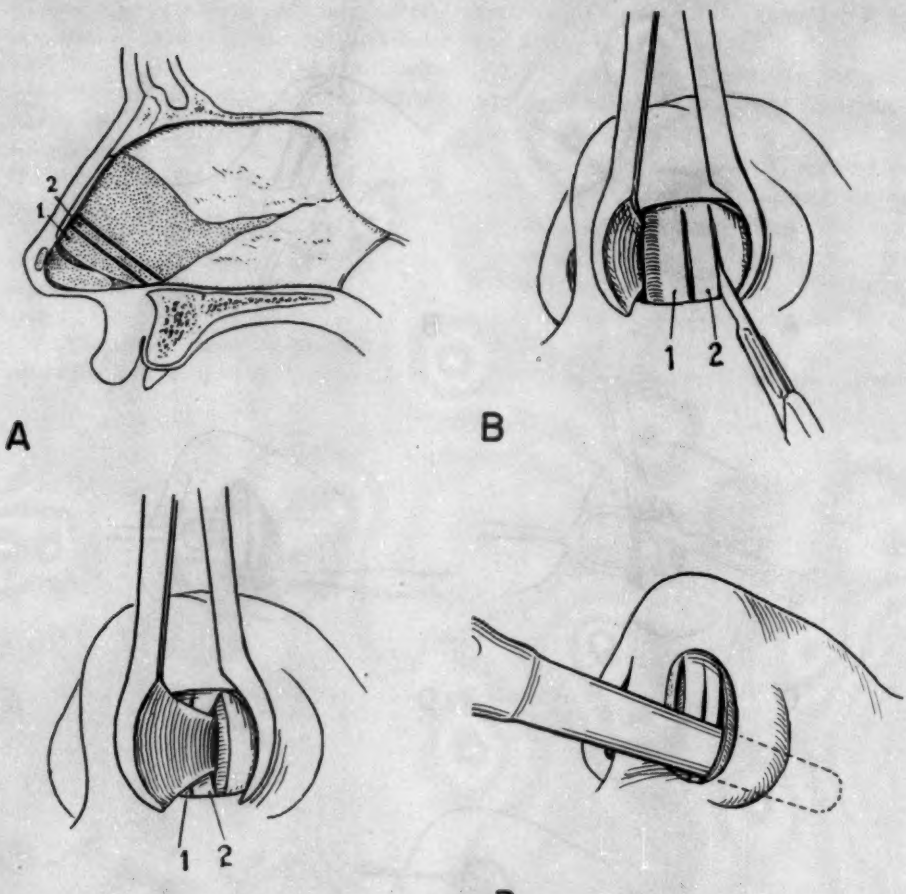


Figure 3

on one side and the tension of the unfreed side was prone to cause recurrence of the deflection. The mobilized septal segment had to be firm and straight in order to be replaced in the midline. The presence of an uncorrected deviated anterior nasal spine upon which the swung caudal segment rested prevented it from maintaining the reset position.

The next significant advance in septal surgery was made by Fomon. The septum was completely resected and replaced in part. This step was based on a "tie-in" of the upper lateral cartilages with the septum. Fomon believed that the former structures alone could support the cartilaginous nasal dorsum because they were an integral part of the septum with which they were fused and were attached subjacently to the nasal bones and the frontal process of the maxilla. They were thus able to furnish adequate stability to maintain profile projection under static conditions. Nevertheless, after removal of the cartilage he reinserted strips of resected cartilage only to prevent secondary cicatricial contraction and thus avoided an external dorsal depression.

It was found, however, that after the septum had been completely removed a "saddle" occurred, which subsequently became

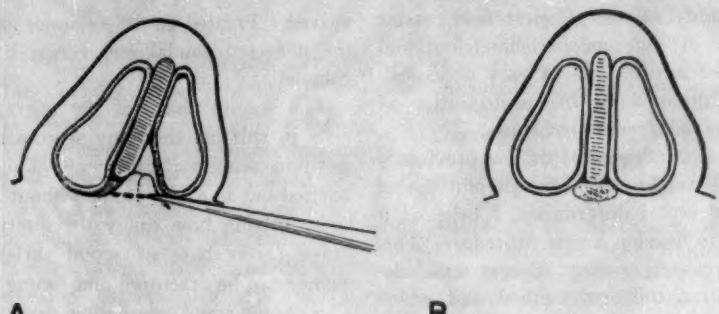


Figure 4

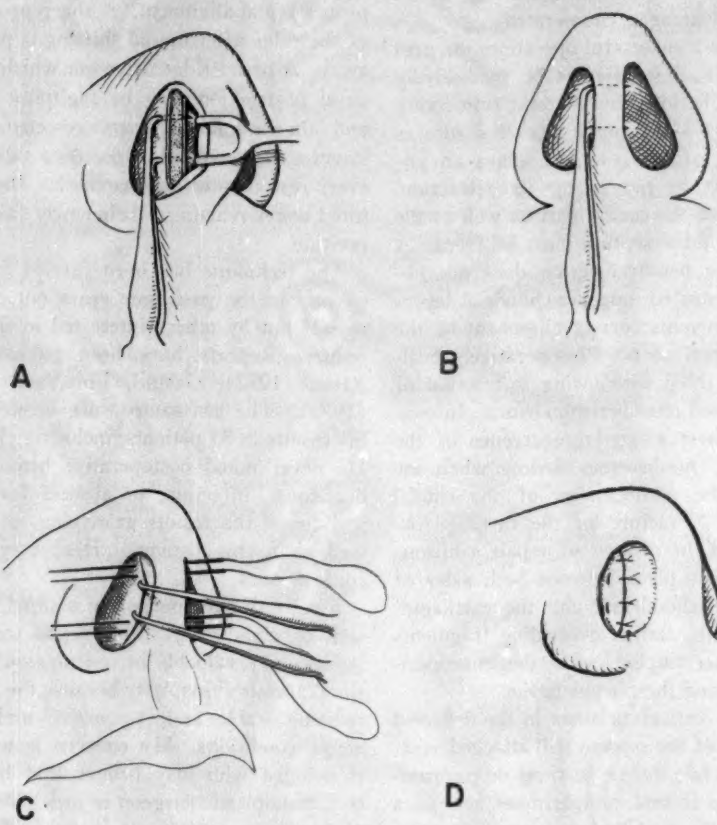


Figure 5

more pronounced. The cartilaginous implants between the mucoperichondrial layers frequently became twisted and often to a degree that they caused nasal obstruction.

Despite the inadequacy of the operation, it was a distinct step in the right direction. Fomon deserves a great deal of credit for emphasizing the significance of the anatomy and physiology of the septum and upper lateral cartilages. He popularized the rhinoplastic approach for septal resection by showing its potentialities—ease of approach,

clear visibility of the surgical field, easier separation of the mucoperichondrium and mucoperiosteum because of easy accessibility—thus diminishing the probabilities of perforating adherent membranes.

Owing to inadequacies of the previously mentioned techniques, although still useful in selected septal deformities, I believed it advisable to develop a new procedure. This would adequately manage diverse septal deviations in a uniform method and which would not be handicapped by the possible limitations of those mentioned but would have the advantages enumerated.

To ensure a successful operation, the previously outlined steps must be successively followed. The low quadrilateral transfixion with through-and-through mattress sutures at the base of the columella plays an important part in preventing its retraction. Both sides of the caudal part as well as the remainder of the septum must be freed. A unilateral or hemitransfixion does not liberate the distorted mucoperichondrial layers and thus prevents correct alignment of the replaced septal parts. Postoperatively healing is interfered with owing to traction of the unelevated mucoperichondrium. In consequence, there often is recurrence of the deformity. This becomes obvious when one considers the pathogenesis of the caudal deformities. Fracture of the cartilage occurs, and in the process of repair a fibrous invasion takes place between both sides of the mucoperichondrium and the cartilaginous fragments, and/or overriding fragments become intermingled with the mucoperichondrium and the fibrous tissue.

The two vertical incisions in the deflected caudal part of the septum still attached ventrally create two mobile sections or compartments. The second compartment acts as a buttress to the first and prevents its retraction. The caudal end of the septum may be very thin or, in the extreme example, purely fibrotic without resemblance to cartilage. Partitioning and making sections, even in this kind of tissue, eventually induces rigidity for ample support. If the caudal end of the septum is unusually thick it can be shaved. Treated in the manner described, the deflected caudal end forms a straight columella.

The second phase of the operation consists in shifting the bony septum into midposition with a "displacer." This step is carried out in a low plane without force. It is surprising how easily the intermaxillary ridge (bony base of septal skeleton) and vomer can be fractured and swung mesially. Overlapping and angulating cartilages over the newly placed ridge are excised to permit proper septal alignment. A sharp projection of the ridge after mesial shifting is punched away. A broad ridge of vomer which causes nasal obstruction may be sagittally spliced and the redundant parts resected. This leaves a narrow nasal ridge. As a rule, however, resection is not required. The fractured bones remain in their newly fashioned position.

The technique has been carried out frequently in the past four years not only by myself but by others interested in the procedure. Reports have been published by Xirau (1952), Galanti, Fine, and Jennes (1954). The last-named surgeon described his results in 33 patients, including children. He never noted postoperative hemorrhage, hematoma, infection, or abscess formation and found the results gratifying to him as well as to his patients. His observations confirm ours.

Finally, I have purposely omitted discussion of rhinoplasty. This septal technique is especially valuable in conjunction with a simultaneous rhinoplasty because the septum remains stable and supportive under dynamic conditions. My concern is with the rhinologist who may believe that he must be a rhinoplastic surgeon in order to accomplish this operation. It is not difficult to learn or be trained in the rhinoplastic approach, which for practical purposes requires transfixion of the septum. Once this step has been carried out the surgeon accustomed to his previous standardized submucous resection can proceed with the advocated septal repair.

**Summary**

A technique is described for the correction of the deviated nasal septum. It is aligned centrally without, or with minimal, sacrifice of structures. The procedure is based on the principle of mobilization of the caudal end of the septum to the midline by utilizing pedicle flaps of the caudal deflections and central replacement of the deviated posterior osseocartilaginous parts. The technique has all the enumerated advantages.

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# Scleroma

## A Review with Report of a Case

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Rhinoscleroma was originally described by von Hebra [1] and Kaposi [2] between 1870 and 1874. One of these authors claimed, however, to have observed a case in Paris some time previously, which in retrospect, he considered an instance of the same disease. Von Hebra and Kaposi considered the lesion a so-called "granulation sarcoma." Later Geber [3] regarded it as a specific granulation of unknown cause. This theory was also championed by Mikulicz,[4] who first studied the lesion histopathologically and after whom the characteristic foam cell was named. In 1882 von Frisch isolated a diplobacillus from the nasal secretions and affected tissue, to which he attributed the disease. Koch's postulates, however, have never been satisfied, and this organism is by no means universally accepted as the specific cause of the disease.

In view of the fact that relatively few cases have been reported in the United States, we feel justified in adding another case to the American literature.

### Report of Case

A Jewish factory worker, aged 50, entered the hospital on Dec. 25, 1953, complaining of difficulty in breathing. The patient was born in Russia and had lived for many years in Cuba. For the past 10 years he had complained of increased difficulty in nasal breathing. Physical examination showed a deviated septum dislocated into the left naris, with almost complete obstruction. The remainder of the

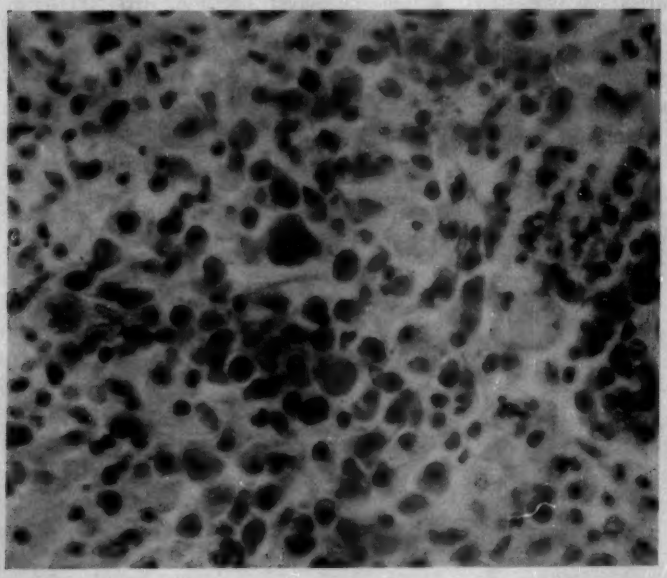

Fig. 1.—There is a pleomorphic chronic inflammatory infiltrate, plasma cells predominating.

Accepted for publication April 30, 1956.

Read before the Rhode Island Pathological Society, Dec. 15, 1955.

From the Departments of Pathology and Ear, Nose, and Throat, Roger Williams General Hospital.

physical examination was negative. A submucous resection and right ethmoidectomy were done, and the patient was discharged as improved.

On May 25, 1955, the patient again entered the hospital, complaining of obstructed nasal breathing and left-sided epistaxis. This condition had lasted for about three months. Examination showed a small sessile growth on the anterior left septum, about the size of a small pea.

Pathological examination: The specimen consisted of a small, lobulated, mottled purplish-pink piece of tissue measuring 8×7×1 mm.

Microscopic study showed an ulceration of the overlying stratified squamous epithelium, and where present the epithelium was not remarkable. The stroma immediately beneath was the site of a dense, pleomorphic, chronic inflammatory infiltrate, made up of many plasma cells, lymphocytes, and

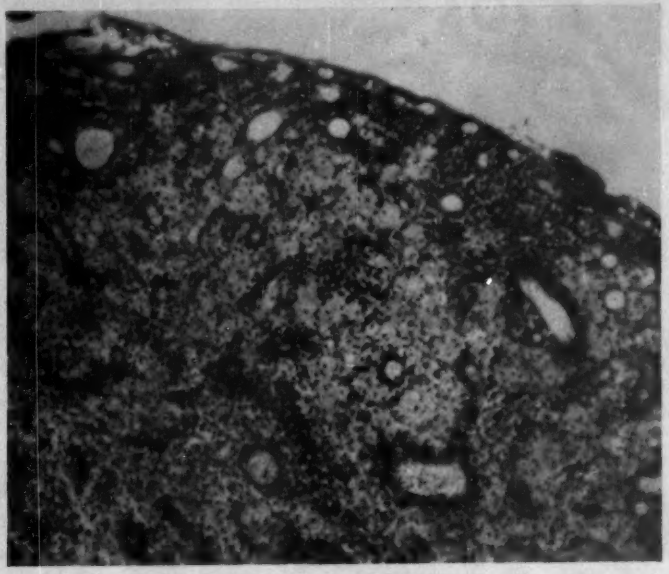

Fig. 2. — Myriads of pale Mikulicz cells monopolize the microscopic field.

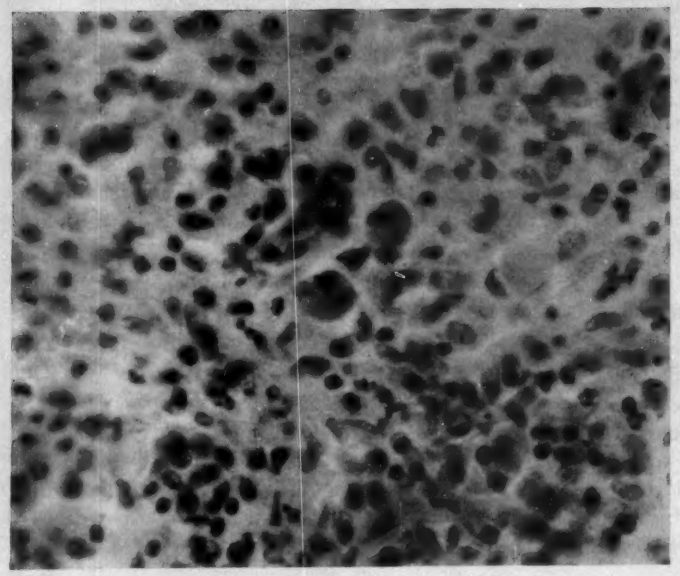

Fig. 3. — Large centrally located Russell-Unna bodies are surrounded by acute and chronic inflammatory cells.

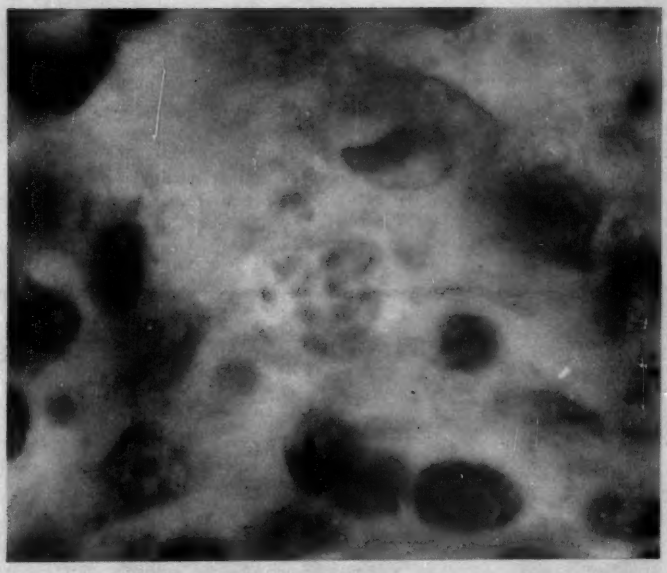

Fig. 4.—A small cluster of K. rhinoscleromatis is found in a Mikulicz cell.

numerous small aggregations of polymorphonuclears (Fig. 1). Scattered throughout this infiltrate, singly and in groups, and forming a very conspicuous feature of the entire ensemble, were numerous large, foamy, pale mononuclear histiocytic cells of Mikulicz (Fig. 2). Occasional large ovoid to round, intensely eosinophilic masses without nuclei or with pyknotic nuclei, the Russell-Unna bodies, were present (Fig. 3). A few fibrotic areas were present, but fibrosis was not a conspicuous part of the case.

Sections stained by Giemsa's method revealed, in many of the Mikulicz cells, particularly in those near the surface of the lesion, small rod-shaped encapsulated organisms, regarded as the von Frisch bacillus or Klebsiella rhinoscleromatis (Fig. 4). No acid-fast organisms could be demonstrated.

A diagnosis of scleroma (rhinoscleroma) was made, and the diagnosis was confirmed by several other pathologists.

Recovery was uneventful, and the patient was discharged as improved.

## Comment

This interesting lesion is very rare in the United States and Canada. One of the most instructive papers in the recent literature is that of Cunning and Guerry,[5] who in 1942 reviewed the literature to date, presenting a most instructive and comprehensive résumé of the subject. Including their own cases, they found a total of 102 reported cases in the United States and Canada, of whom 16 were native-born.

Rhinoscleroma is an extremely chronic granulomatous lesion involving the respiratory tract, and specific involvement has been reported of the septum, skin of the upper lip, cheek, gums, antra, lachrymal ducts, Eustachian tubes, nasopharynx, hard and soft palate, tonsils, pharynx, larynx, trachea, and bronchi to the smaller ramifications. In view of this widespread potential involvement, in 1932 the International Congress of Otology, Rhinology, and Laryngology, in Madrid, adopted the more inclusive term, "scleroma," as the standard designation of the lesion.

In scleroma the affected tissues, either diffusely or discretely, become indurated and cartilagenous in consistency, and, after a variable length of time, fibrotic. The disease is apparently limited to the skin and mucosa, but the end-result may be the appearance of most grotesque and horrible deformities. Involvement of the lower respiratory tract may result in distressing dyspnea, or even in death by asphyxia. To the sectioning knife the involved tissue is

hard, difficult to cut, and cartilaginous in consistency.

In most cases a characteristic microscopic picture is seen. The lesion consists essentially of a granulomatous infiltration followed by marked fibrosis. The epithelium is seldom ulcerated. It may be atrophic, or hyperplastic even to the production of pseudoepitheliomatous hyperplasia. The infiltrate is composed chiefly of plasma cells and lymphocytes. Throughout this infiltrate one sees numerous Russell bodies, rounded bright red cells sometimes containing a pyknotic nucleus. These bodies probably represent degenerated plasma cells and are nonspecific. Mikulicz cells are more specific, and are large, foamy, vacuolated cells, probably of histiocytic derivation, which contain a small nucleus. They are found in greatest numbers near the surface of the lesion. They do not contain fat. In many of the Mikulicz cells the characteristic von Frisch organisms (K. rhinoscleromatis) can be demonstrated by appropriate staining methods. Fibrosis is usually encountered and in late cases it may be quite marked.

The symptoms are quite variable, depending on the area of involvement and the extent and duration of the lesion. The process usually begins on the nasal septum and is accompanied by a foul discharge, crusting, and epistaxis. Later the nares may be completely blocked by granulomatous masses, and the nasal architecture may be so distorted, and mutilations may be so severe, that the features of the patient become almost unrecognizable. Other symptoms include hoarseness, aphonia, brassy cough, dyspnea, stertorous breathing, ozena, deafness, tinnitus, and sinus headaches. There are no constitutional symptoms.

This disease has always shown certain endemic foci, but in recent years, probably owing to wars, increased travel, and economic dislocations, it appears to have become ubiquitous. Originally the disease was described principally in Egypt, Russia, Poland, and Central and South America.

Cunning and Guerry[5] list the birthplaces of foreign-born cases of scleroma reported in the United States and Canada as follows: Hungary, Galicia, Russia, Austria, Bohemia, Poland, Italy, Mexico, Ruthenia, Ireland, Germany, Roumania, England, San Salvador, and Guatemala. Kline and Brody[6] also mention cases originating in Asia, Africa, India, Siberia, Sumatra, Sweden, and Australia.

The birthplaces of the American-born cases mentioned in the article of Cunning and Guerry[5] are the following: New York, Buffalo, Arkansas, North Carolina, Cincinnati, Baltimore, Trenton, N. J., Michigan, Spartanburg, S. C., Kansas, California, Greenville, S. C., Perth Amboy, N. J., and Brooklyn, N. Y. An endemic focus is believed to exist around Spartanburg, S. C.

It has been a universal observation that scleroma is a disease found almost exclusively in the lower socioeconomic group.

The etiology of scleroma is still a controversial matter. Originally the von Frisch organism was implicated, owing to the fact that its presence could always be demonstrated in the lesions. However, Koch's postulates have never been satisfied and some observers have reported the isolation of this organism from normal nasal secretions. Levine, Hoyt, and Peterson[7]; O'Hara, Pratt, Levine, and Hoyt[8]; Levine and Hoyt,[9] and Levine[10] have presented further evidence implicating K. rhinoscleromatis as the cause of scleroma. According to these investigators K. rhinoscleromatis possesses a definite biochemical pattern exhibited by no other Gram-negative rod. They claim that this organism is never found in normal noses and is never found in conditions other than scleroma. They regard the isolation of K. rhinoscleromatis as diagnostic of scleroma.

Diagnostically, scleroma must be clinically differentiated from syphilis, tuberculosis, leprosy, neoplasms, infective granulomas, chronic glanders, atrophic rhinitis, Boeck's sarcoid, and verrucous leishmaniasis.

The definitive diagnosis is made by

biopsy. There is usually a preponderance of plasma cells, perivascular in distribution. Plasma cells in conjunction with the Mikulicz cells and the Russell bodies are highly suggestive. Mikulicz cells, however, have been observed in leprosy, glanders, bubonic plague, and granulation tissue of wounds. Russell bodies have been seen in tuberculosis, syphilis, leprosy, chronic infective granulomas, some malignant tumors, and some diseases caused by filtrable viruses.

However, a granulomatous lesion from the respiratory tract showing plasma cells, Mikulicz cells, and Russell bodies, in which K. rhinoscleromatis can be demonstrated and/or isolated, and in which no acid-fast organisms can be found, is almost certainly scleroma.

An attempt can be made to culture K. rhinoscleromatis. Isolation of the organism is significant whether or not one believes that it is the actual cause of the disease.

Levine and Hoyt [9] have reported encouraging results diagnostically with a complement fixation test using K. rhinoscleromatis as an antigen.

Finally, an intradermal test has been devised using a suspension of killed K. rhinoscleromatis.

The prognosis of the disease is fair as to longevity, but must be guarded as to local distress and the appearance of deformities.

Treatment in the past has not been very successful. Until quite recently the best results seem to have been obtained by surgical removal of the granulomatous tissue, followed by irradiation. Recently good results have been reported following the administration of streptomycin [11] and dihydrostreptomycin.[12]

Since the report of Cunning and Guerry listing 102 patients with scleroma observed in the United States and Canada, several other cases have been recorded.

Dixon [13] reported a case of scleroma in a Jugoslav immigrant. Dill [14] added another case observed in a Polish immigrant, and Kellert [15] described this disease in a Polish-born immigrant who had lived in Massachusetts for many years.

Levine, Hoyt, and Peterson [7] presented six cases of scleroma in one family, all of whom were of Mexican descent. In two cases the results of biopsy were conclusive, but in the remaining four biopsy studies were suggestive but inconclusive. In all cases, however, K. rhinoscleromatis was cultured, and the authors felt, in view of these positive cultures, that these four cases were in an early latent stage.

Again Hara, Pratt, Levine, and Hoyt [8] present seven patients from one family. In two the clinical and biopsy findings were conclusive, although in the remaining five cases no diagnostic biopsies could be obtained. Both positive cases were native-born.

Kline and Brody [6] reported a case of scleroma in an American-born woman, born of Austrian parents, and in her brother. Another brother died of symptoms suggestive of bronchial scleroma, but the case was never proved by biopsy.

Other more or less sporadic reports have appeared in the literature. Putney [16] reported a case in a Polish immigrant, and Miller [17] added three cases, of which two were born in the United States of Mexican origin, and of which one was Mexican-born.

Eisentodt [18] observed a case in a Russian-born Jewess, and Hoover and King [19] recorded a case of scleroma in a Russian-born shoe worker. Dwyer [20] saw a case in a miner of Hungarian descent, and New, Weed, Nichols, and Devine [21] reported a case in a white woman born in Siberia and reared in the Far East.

Olson [22] added three cases, two of which were born in Mexico, and one of which was born in New York of Greek origin. Morwitz and Horwitz [12] studied a case in an immigrant from Kiev, Russia.

Finally, Cunning [23] described scleroma in two native-born brothers.

In summation, then, in so far as we have been able to determine, including our own

case here reported, 126 cases of scleroma have been reported from the United States and Canada, of which 98 were foreign-born and 28 native-born.


## Summary

A case of scleroma in a Russian-born immigrant is reported.

The entire subject of scleroma is reviewed, particularly regarding the incidence of the disease in the United States and Canada.

Review of the literature indicates that approximately 126 cases of scleroma have been reported in the United States and Canada. Of these 98 were foreign-born and 28 were native-born.


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# Tinnitus, Deafness, and Vertigo

## A Discussion of Etiology

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Just what do we mean when we use the words tinnitus, deafness, and vertigo? The following brief definitions are submitted to orient our understanding.

Tinnitus is an autogenous sensation of noise * in the ear or in the head.

Deafness, partial or total, even sudden deafness, is a sensation only in the sense of being a lack of sensation, a loss of hearing, real, imagined, or malingered.

Vertigo is a disturbing sensation of turning, unsteadiness, or disorientation of the position or movement of the body in space or in relation to the environment itself or to gravity.

We shall not at this time discuss the anatomy and physiology of the tissues and organs concerned with tinnitus, deafness, and vertigo except incidentally some items under discussion.

There are two distinct kinds of head noise: (*a*) vibratory, which is caused by actual vibrations in the tissues or structures in and about the head or its vascular channels; (*b*) nonvibratory (true tinnitus), which is an illusion of vibration caused by irritations of the auditory neural tissues. In some mental disorders tinnitus may be a delusion or a hallucination. Tinnitus is an objective or subjective symptom, depending upon its type and severity. Except in its vibratory form tinnitus cannot be heard by the examiner. We are herein concerned with it only as an illusion of sensation. It may be measured with considerable accuracy.

There are two fundamentally distinct types of deafness: (*a*) obstructive (impedance) deafness (outer, middle, and inner ear) which is frequently reversible; (*b*) neural deafness (inner ear, nerve, and nuclei) which is not reversible except in its predegenerative stage. Types (*a*) and (*b*) may occur separately or superimposed one upon the other. Deafness may occur as an illusion, a delusion, or a hallucination in mental disorders. We are herein concerned with "neural deafness" and with inner ear impedance deafness. Both types of deafness can be measured with accuracy. Deafness is a subjective symptom, objectively demonstrable and measurable.

There are two distinct types of dizziness: (*a*) physiological, caused by normal reactions to movement of the body or its environments; (*b*) pathological, which is an illusion or false sensation of movement of the body or its environment. Pathological dizziness, or true vertigo, may be (1) linear, a sensation or movement of the body or of the environment in a straight line; (2) rotary, a sensation of movement of the body or of the environment in a circle or curved line. Either may occur separately or be superimposed one upon the other. As in the case with tinnitus, dizziness may be a delusion or a hallucination in certain mental disorders. We are herein concerned with both linear and rotatory types of the illusion.

Dizziness is a subjective symptom, ob-

Accepted for publication April 9, 1956.

Read before the New Jersey Ophthalmological and Otolaryngological Society, Atlantic City, March 17, 1956.

Largely taken from part of Chapter XI, Fowler, E. P., in Otolaryngology, edited by G. M. Coates, H. P. Schenck, and M. V. Miller, Hagerstown, Md., W. F. Prior Company, Inc., 1955.

* According to the American Standards acoustical terminology, "A noise is any undesired sound." However, under some circumstances a noise also may be a desired sound, as, for example, when one wishes to employ it or wants to hear it.

jectively demonstrable and reversible, depending upon its etiology, severity, and place of origin. By clinicians the words "vertigo" and "dizziness" are often employed synonymously. People often use the word, "dizziness" to describe a variety of sensations. A patient may say he cannot walk straight, that he feels as though he was being pullled to one side, or that he is dizzy, wobbly, or unsteady, or that the environment is moving, wobbling, or turning. Vertigo is measurable but with questionable accuracy.

It should be kept in mind that all three of these sensations (tinnitus, deafness, and vertigo) are symptoms, not diseases or lesions per se. They may occur singly or in various combinations with one another and with other associated symptoms, and in varying degrees and patterns of sequence, constancy, duration, and repetition. When without known cause they occur together in attacks, the triad is known as "idiopathic hydrops of the labyrinth," "endolymphatic hydrops," "Ménière's symptom complex," so-called "Ménière's disease," "Ménièriform symptom complex," or "neurovascular labyrinthosis." (The emphasis originally was on the vertigo, nausea, and deafness.) When one or two of the triad are absent, the term "pseudo-Ménière's" is sometimes used. The necessary degrees and combinations of the three symptoms and the etiology necessary to warrant these terms are still uncertain. We do exclude the term "Ménière's" when the symptoms are caused by suppurative disease of the labyrinths.

I agree heartily with H. L. Williams when he says, "When Ménière's papers are read, the reader is forced to conclude that Ménière had a better insight into the disturbance now named after him than many of his otologic descendants have had."

## Etiology

Whereas heredity alone is not sufficient to initiate disease, a favoring environment being also a prerequisite, heredity certainly sets the stage and prepares the actors (the tissues and organs) for the play they are to enact during their lives.

A fair guide for suspecting a hereditary factor in a disease or disorder is its occurrence (1) in two or more instances in the same generation or in different generations of a family; (2) in several siblings of a consanguineous mating; (3) in both of a pair of identical twins.

There is often a family and personal history of migraine in patients with Ménière's. Recurring attacks of tinnitus and vertigo or of deafness or of any two or all three of these symptoms indicate unstable vasomotor control. Psychosomatic and somatopsychic instabilities certainly tend to run in families.

Some physicians consider "Ménière's disease" a primary affection of the vestibular apparatus, and some (the majority) believe it results from a vestibular reaction to a constitutional disorder. The symptoms may in fact be caused by any imbalance or bilaterally unequal disorder of any part of the neural mechanisms of the auditory and vestibular tracts. The symptoms are often accompanied by visible vasomotor phenomena.

In the white race the incidence of "Ménière's symptom complex" as first seen by the physician is highest between the years 40 and 60. In varying degrees it is an affection of the cochlea as well as of the vestibule. In adolescent girls the incidence is low, with a tendency to appear greater near and immediately after puberty. In young boys or young men it is rarely encountered. It may be a significant observation that in youth the tissues generally, and the blood vessels in particular, are more elastic, and the muscles in their walls more mobile and more normally responsive to stimuli than later in life. The incidence in Negroes is extremely low. Climate and habits of living appear to be factors, and similar treatments appear to vary in efficacy in different countries.

The fundamental causes of tinnitus, deaf-

ness, and vertigo may be, and often are, similar, but they may be wholly dissimilar, and the symptoms may reflect more an unhealthy or constitutionally highly sensitive neural apparatus than one primarily affected at the time of their onset. The etiology is often undeterminable either clinically or at autopsy. It may be said that in general these symptoms may result from direct or reflex interference with the mechanical or biochemical functioning of the neural elements anywhere in the right or left peripheral mechanisms of the ear, or from irritations or degenerations anywhere along the auditory and vestibular pathways. The sudden onset of the vertiginous symptoms or differing combinations of the symptoms in typical attacks is due to bilaterally unequal sudden, or rapidly changing, stimulations (or depressions).

Both divisions of the eighth nerve are so highly sensitive that it is often impossible to detect or measure the stimuli involved in their threshold excitation. The sensitiveness of the cochlea and the vestibular end-organs is of the same order of magnitude. Slight changes in environment, in pH or excess salt intake, or in mineral content of the tissue juices (the inter- and intra-cellular fluids), also acoustic shock, pressure, fatigue, degeneration or regeneration, anoxic, toxic, or traumatic lesions are frequent initiating or exciting factors.

With the patient standing in the Romberg position a slight increase in the air pressure in the external auditory canal may even in the absence of any disease, cause the head to deviate towards the opposite side. A caloric change of a fraction of a degree or a loud sound stimulation, by upsetting autonomic balance, may produce comparable effects. Frequently involved are indirect and reflex effects from emotional episodes, disorders of the endocrine glands and of the autonomic neural mechanisms of the body. In fact emotional (neuropsychologic) and vasomotor instability are major etiologic factors, and especially the trigger factors in most patients, although it is sometimes difficult to recognize the psychosomatic interrelationships. Even in the absence of any peripheral disturbing sensation or in the absence of any dysfunction in the brain stem, variations in cortical stability or excitability can cause dizziness. The neural mechanisms of the ear are excitable from vascular subcortical as well as cortical neural reactions. Spasm, contraction, congestion, dilatation, blocking of the vascular channels, and edema from any cause are commonly involved in the mechanisms of disturbed function.

Any interference with the proper functioning of the circulation, or even an abnormal vessel course, may make conditions favorable for anemia, local anoxia, and interference with the nutrition of the neural tissues and cause various disturbing side-effects. The faulty liberation of histamine-like substances will add to abnormal histamine sensitivity, and all or any one of the above factors may alter the permeability of the labyrinth membranes and capillary walls, with a resultant edema, pressure, anoxia, malnutrition, and frequently an ultimate neural degeneration. It is my opinion that increased retention of sodium and other electrolytes, and water retention from any cause, in some patients, are capable of setting the stage for so-called hydrops of the endolymph. Hypothyroidism and some of the other endocrine disorders are conducive to water retention.

A considerable number of patients suffer from subclinical or threshold forms of nephrosis as a phase of undiagnosed chronic glomerulonephritis. Nephrosis causes edema in various parts of the body, usually so slight that it is not noticed or is ascribed to other disorders. Edema is largely an increase in the extracellular fluids. Hydrops also is an extracellular increase in fluid brought about by local and general factors, all of which should be kept in mind. It is well known that a restricted salt intake (1 to 2 gm. a day) diminishes edema in these nephrosis patients. However, it does not alter the course of the disease. It would

seem that a restricted salt intake also would at least temporarily tend to diminish hydrops of the labyrinth when it can be ascribed in part or even primarily to lesions causing irreversible kidney disorders.

In every part of the body the control of the flow of the blood and tissue fluids into and out of the capillary beds depends upon the local capillary permeability, and the opposing actions of the capillary and arteriolar blood pressures and the plasma colloid osmotic pressures. The diameters of the arterioles and capillaries and their pressures are constantly changing and are acutely responsive to bilateral and unilateral sympathetic irritations or stimulations, and emotional instabilities.

Anaphylactic shock has been proved to cause acute vestibular imbalance, but its exact mechanism has not been explained. Allergy is commonly present in patients with Ménièriform episodes (in some locations about 30% of the cases), and appears capable of supplementing, but alone has rarely been observed to cause the typical attacks. The same may be said of oronasal infections.

Certain drugs and chemicals in the blood stream appear to have a selective action on the auditory and vestibular mechanisms, as do other foreign and pathological substances. By selective action is meant a preponderant but not necessarily an exclusive effect upon the organs in question. Many persons have an idiosyncrasy to quinine, tobacco, the salicylates, streptomycin, the sulfonamides, arsenic, salvarsan, the barbiturates, morphine, mercury, gold, alcohol, and many other commonly employed medicines. These drugs are said to have a toxic effect upon the end-organ, spiral ganglion, nuclei, and nerve fibers. In sufficient amount they affect the blood cells and plasma. Their effect may be direct because of arteriole and capillary wall damage, or indirect because of a reflex dilation or contraction of the vessels, thus directly or indirectly changing the permeability of the vascular walls and interfering with nutrition, oxygen supply, and the endolymphatic pressure.

Severe poisoning will cause complete degeneration of the neural tissues with total loss of function. Mild poisoning may cause only temporary changes in the consistency or quality of the perilymph and endolymph and irritative effects such as mild tinnitus, vertigo, or deafness, or only one or two of these symptoms. Conditions short of neural degeneration are frequently reversible. It is pertinent to observe that in mild poisoning the effect of most of these drugs is more on the auditory than the vestibular end-organ, which accounts for the fact that the first symptom of poisoning is usually a tinnitus, followed by a lowering of the hearing. Usually only gastrointestinal irritants or severe poisoning cause vertigo, nausea, and vomiting. Tobacco, alcohol, and streptomycin appear to have no special monopolizing affinity for the tissues involved in equilibration, but they certainly are potent causes of dizziness. Toxic effects from these poisons may persist and accumulate for weeks even after they are no longer taken.

Episodes of vertigo are caused by disturbed coordinations between the many factors regulating the balance in postural attitudes of the body whether at rest or in motion, and especially in the static labyrinth, muscle and joint tonus, vision, and the kinesthetic mechanisms. As I see it, the trigger is pulled by sudden changes in pressures or sudden traumas (bodily or mental), not by gradual changes and not during stabilized states. Therefore rapid changes in the rate of or direction of movement of the head or of the body, or of either in relation to the other, and cardiovascular, digestive, and psychogenic disturbances are common instigators of dizziness.

Although many of the neural pathways are unknown, it is known that there are numerous connections between the auditory and vestibular nuclei, and other brain areas.

Disease of the central nervous system, particularly if it involves the posterior

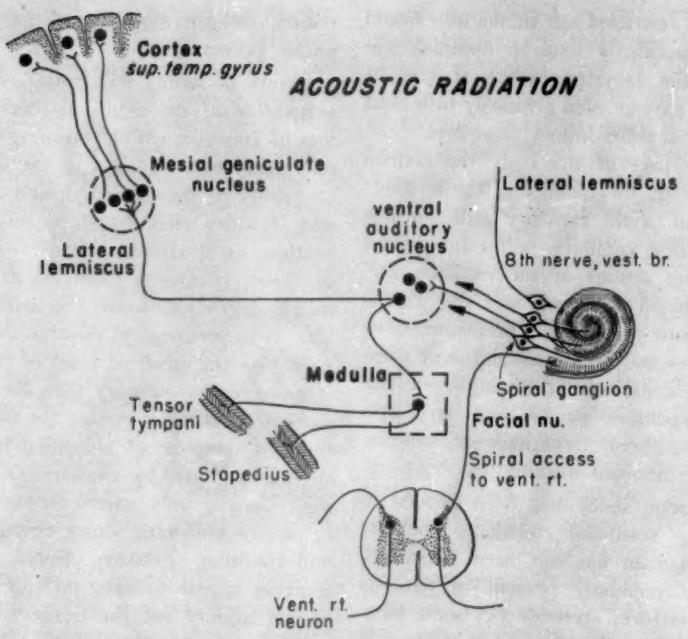


Fig. 1.—Diagram of the principal neural connections of the auditory nuclei modified from Lorente de Nó. The central auditory pathways provide channels for reflex as well as for direct hearing impulses; also for the tinnitus impulses. The latter may at times be related to the "delay circuits" through the reticular substance of the pons.

cranial fossa, often causes various combinations of tinnitus, deafness, and vertigo. Real dizziness is very disturbing and therefore always accompanied by a definite excitation of the vegetative nervous system, and it is conducive to a general lack of well-being. On the other hand, physiological stimulation of the vestibular apparatus is as a rule transient and not disturbing. A summation of stimuli or accelerating stimuli acting simultaneously or in quick succession in several planes causes a much greater excitation of the vegetative system, and a greater and more disturbing sensation of dizziness than stimuli active in one plane.

Either hypertension or hypotension may contribute to tinnitus, deafness, and vertigo, not only because of the abnormal blood pressure per se, but because in either of these states there are apt to be rapid bilaterally unequal changes in the pressure from time to time, and these pressure changes are transmitted to the labyrinth capillary beds and fluids. In some persons even blowing the nose or gentle Politzerization may cause vertigo and a temporary deafness; also blockage of the Eustachian tube from barometric changes during flying or from diving under the surface of the water, or from any type of stenosis. The sudden relief of an abnormal pressure in the middle ear may cause vertigo. Even normally respiration, by its effect upon the vascular channels in the ear, exerts a slight pull and push effect on the labyrinthine fluids. Not only the cochlear aqueduct but also the ductus saccus and endolymphaticus may be affected by these changes in pressure.

Variations in the lymph fluids of the labyrinth may be caused by intermittent or chronic lesions in the middle ear, the blood stream, or the cranial cavity. Toxic poisoning, bone dyscrasias, electrolyte, endocrine, metabolic, and pressure lesions of various kinds can cause anoxia of the neural fibers, the ganglia, and the end-organ elements in one or both ears. Oseoarthritic changes, and particularly with spondylosis of the

cervical spine, may be a factor causing vertigo because neck traction or trauma may act as the trigger mechanism.

Clinical experience shows that an increase in intracranial pressure from hydrocephalus or tumors rarely causes labyrinthine symptoms while the subject is in a horizontal position, but changes in position if sufficiently sudden, may cause a transitory deafness, tinnitus, vertigo, and nystagmus and maybe nausea and vomiting, even in normal persons. In such instances even in normal persons there is an element of what we call an emotional reaction. It would appear that it is the sudden change in hypersensitive or autonomically unstable individuals that pulls the trigger to start the mechanism of Ménièriform attacks, as well as of certain attacks and types of tinnitus and deafness without vertigo.

or tonic contractions of the blood vessels with resultant anoxia in the tissues affected. It should be kept in mind that quinine, morphine, alcohol, and other drugs, as well as some of the viral, spirochetal, and bacterial diseases (rubella, syphilis) in the mother can pass to the fetus by way of the placenta, or to the nursing baby by way of the breast milk. However, in moderate therapeutic amounts quinine does not appear to be a serious hazard unless there is an idiosyncrasy in mother or child. A diagnosis of hereditary deafness should not be made without eliminating the possibility of poisoning by way of the placenta blood or the breast milk.

The salicylates are usually well tolerated, but persons with an idiosyncrasy to them may develop severe symptoms of irritation and even of degeneration of the auditory and vestibular organs. Such symptoms are

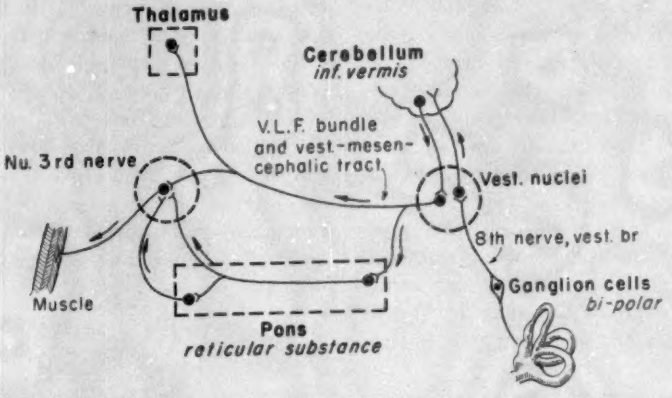


Fig 2.—Diagram, according to Lorente de Nó, of the principal connections of the vestibular nuclei in the pons, the cerebellum, and the thalamus. De Nó emphasizes the importance to the vestibular mechanism of the role of the "delay circuits" through the reticular substance of the pons. These may set up reverberating circuits capable of prolonging or augmenting vertigo.

There is a linkage of the fifth nerve to the vestibular analyzer mechanism, and it is therefore probable that atypical labyrinthine reactions in Ménière's may be caused by conditioned reflexes set up via the fifth nerve.

Quinine is a protoplasmic poison and has a profound effect upon the pulse, the blood, and the vessel walls. It may cause clonic

manifested by tinnitus (often loud), vertigo, and deafness, and sometimes in addition by other sensations. The symptoms fade if the salicylates or other drugs are discontinued before permanent damage results, but in event of tardy withdrawal symptoms may continue for a long time and even remain permanently.

Quinine, the salicylates, and other poison-

ous drugs should be used with caution because, although the patient may react but mildly, if continued such drugs may gradually weaken or destroy the more susceptible neural elements, so that subsequent added disease, trauma, or circulatory disorders will more easily irritate or destroy them in whole or in part. Once damaged, the auditory and equilibrium mechanisms will be less resistant to subsequent insults from any source.

Large doses or prolonged treatment with streptomycin usually causes a toxic effect more marked in the vestibular portion of the eighth cranial nerve, while dihydrostreptomycin affects more the auditory portion.

Alcohol and tobacco are pleasant poisons, and they may and sometimes do contribute to tinnitus and deafness, but we are seldom able to prove it. However, both of these poisons loom large in the etiology of vertigo; it is probable they affect not only the end-organs in the cochlea but also the cerebellum and cerebrum. At first, like all posions, they stimulate and then they depress the circulation. Many people develop a tolerance to these poisons, and seem immune to their damaging effects for years.

Carbon monoxide and disulfide, the aniline dyes, lead, phosphorus, and many other poisons used in industry, are capable of causing tinnitus, deafness, and vertigo of varying degrees, according to idiosyncrasy, the amount ingested, and the time involved.

Generally speaking, blood dyscrasias, anemia, leukemia, and depletion or loss of blood from any cause frequently produce tinnitus, less frequently true vertigo, and still less frequently neural deafness. There appears to be an idiosyncrasy for these conditions, because patients have been observed with severe pernicious anemia (25% Hb.), and almost in extremis from other blood dyscrasias, without sensing any tinnitus or vertigo, and even careful audiometric testing revealed no deafness in some patients who died within a day or so subsequent to the testing.

Blood sludge has been observed in many instances coincidentally with disorders of the ears, and in my opinion it is intimately involved in the etiology of tinnitus, deafness, and vertigo. It regularly accompanies infections, intoxications, sympathetic nervous system instabilities, blood dyscrasias, spasms of the medium and smaller vascular channels, and local edema from any cause. The capillary beds before menstrual periods are engorged, and there is an increase in water and electrolytes in the body with sludging of the blood with these episodes. Sludging of the blood is also observed incident to childbirth and the menopause. Its presence temporarily appears to be a defense mechanism, but when prolonged or often recurring it may be a menace to health. It is less prevalent in children. The difficulty is to determine when and to what degree the sludging of the blood and the accompanying phenomena are and when they are not important factors in the production of the symptoms and the coincidental pathology.

Blood sludge is present locally and generally in practically all of the conditions which originate from irritation or reflex stimulations of the sympathetic nervous system. It may be most easily observed in the arterioles and venules of the conjunctiva by means of a binocular microscope and high magnification under brilliant illumination.

### Blood Sludge

By sludging of the blood is meant the formation in the blood plasma of very minute to very large gelatinous agglutinated lumps or masses of red blood cells of varying lengths and diameters, depending largely upon the diameter of the vascular channels into and through which they are being squeezed.

In healthy, conscious, and relaxed humans and in healthy lower animals the circulating erythrocytes tend to keep separate, to slightly repel each other. Rouleaux are not found. There seems to be no tendency to adhere or stick together, or to adhere to the smooth walls, even of the smaller

vessels. There is no agglutination or clumping of the cells in health,[1] unless the intravascular agglutinating mechanism is set in operation by some poison, trauma, shock, physical or psychic, fear, frustration, or hypersensitivity to autonomic repercussions. All of these affect the endocrine balance.

The healthy normal flow of the blood is laminated concentrically, the center stream of corpuscles in the plasma traveling most rapidly, and each successive surrounding layer or stream (each one red cell thick) traveling slower and slower as the layers approach the periphery, the slowest moving layer being that next to the intima of the vessel wall. Minimal internal friction is thus obtained for the passage of the blood in arteries and veins, and the most rapid possible rate of flow which is available throughout the successive pressure drops in each branching and narrowing along the course of each patent vessel and its branches.

The terminal vascular channels constitute a perpetual bottleneck in the circulation. However the blood in most arterioles and venules flows so rapidly that "the individual red cells can not be seen," as Knisely [1] put it. A useful criterion for adequate blood flow is in fact the inability to see the individual red cells. It has been shown that little or no blood fluid is being lost through the capillary walls if the speed of the oxygen-saturated arterial blood is so fast that the cells under high magnification cannot be seen in these small vessels.

A fair sample of the circulating arterial blood may be observed by examining the blood in the obliquely illuminated bulbar conjunctival vessels. For this purpose the slit-light apparatus is satisfactory for clinical use and it also enables one to see the small vessels and observe their dimensions, form, and vasomotor stability and responses to stimuli, if sufficient illumination and magnification are used.

Oxygen is one of the most important, if not the most important, constantly needed blood-borne materials. It is not stored in the tissues. Moreover its supply varies from time to time according to the rate of blood flow in each tissue, and the blood flow is largely proportional to the size of the vessels. The narrowest or most completely contractible vessels are the arterioles, the terminal arterioles, and the contractile sphincters, the diameters of which change from moment to moment as demanded by physiological adjustments in oxygen, glucose, and the other materials which the blood brings to the tissues. Also the blood vessels vary in diameter with changing demands for removal of wastes, of heat, or because of the varying requirements of some special and indispensable organ which depends upon the blood it receives to properly function.

Many factors affect the response of the contractile elements involved in blood flow. Almost every whole arteriole-to-capillary, or sinusoid-to-venule channel can contract, even to the extent of completely closing its lumen throughout its entire length. Such a road block forcibly resists the entrance and passage of blood and is always associated with sludging of the blood. On the other hand, individual parts, or all parts, of vessels can dilate to their maximums.

It is clearly evident that all the variations in the smaller vessels and capillary beds must have a profound effect upon the health of the tissues, and upon the hydrostatic balance and the neural health and sensitivity. Whenever one has been fortunate enough to see a patient just before, during, or after an attack of the typical symptoms of Ménière's disease, he will always find considerable sludging of the blood, and frequently in people with other types of dizziness.

Whether or not it and its accompanying phenomena are the principal cause of the attacks, the presence of sludge is an indication of faulty metabolism and a cause of anoxia due to plugging of the vessels in somewhat the same way as with small emboli.

In fact, sufficient slowing of the blood in the small vessels will instigate intravascular coagulation by subjecting the blood cells

to squeezing and scraping along the endothelial lining, thus bruising it and changing it into an abnormal tissue. Any abnormal or foreign tissue engenders agglutination or coagulation of the blood, depending upon quantity and the extent and speed of the environmental changes. Agglutination causes slowing of the circulation with an increased permeability in the capillary beds, with anoxia, congestion, stasis, edema, extracellular hydrops, and some or all of its sequelae.

I again stress the point that it is not necessarily the hydrops that causes the degenerations or even the symptoms in Ménière's disease; it is rather the lesions which cause the hydrops. They are the culprits. It is the neurovascular phenomena with their pathophysiological effects, and the accompanying autonomic and somatopsychic repercussions which are at the base of the trouble.

This opinion is supported by the fact that at autopsy hydrops of the labyrinth may be found without any history of Ménière's disease, and vice versa. What I am trying to say is that the presence of the hydrops alone does not necessarily appear to be the cause of the disease or of the symptoms. The pressure it exerts is distributed equally to all of the cells and cellular tissues endure equally distributed pressures remarkably well unless the circulation is insufficient for good health. But the moment circulation falls to the critical threshold of insufficiency, then the trigger is pulled, and off goes the explosion, and often the subsequent rapid or slow degeneration of the neural tissues.

The degree that sludging of the blood per se can cut down oxygen supply is not generally realized. Even moderate intravascular coagulations can deprive the tissues of more than half of their normal supply of oxygen; cutting down the availability of the metabolites without question adds insult to injury by an incomplete removal of wastes.

From long experience I have learned that most medical men are not so much interested in etiology as they are in what particular medicine, or what particular surgery, is indicated to, as they say, "cure the disease." Now, strictly speaking, no medicine and no surgery cures a disease. They may alleviate symptoms, they may extirpate an organ, they may to some degree restore continuity of tissues and eradicate pus, but they do not cure. Both medicine and surgery are but aids to help the patient cure himself.

Without some understanding of the etiology of a disorder or a disease, the physician is ill equipped to properly treat it, and this is the reason that I have taken your time and mine to discuss the etiological factors in tinnitus, deafness, and vertigo. I trust I have been able to give you something to think about so that neither you nor I will have given our time in vain.

140 E. 54th St.

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# Surgery of Carcinoma of the Larynx

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The complete removal of the primary growth is of the utmost consideration in any operative procedure. The general physical condition of the patient must be such that he is a reasonably good operative risk. There are so many individual problems one could bring out in surgery of carcinoma of the larynx that to cover the subject in a short article is well nigh impossible. I have taken some selected problems pertaining to the classification and treatment of cancer of the larynx rather than delve into a more scientific discourse.

This paper is based upon a series of 623 cases of cancer of the larynx seen in my private practice. Fifty-nine of these patients were inoperable at the time of my first examination. Eighty refused any operation. Forty-three patients had other treatment, such as deep x-ray therapy. This leaves 441 cases of cancer of the larynx operated on, which forms the material for this presentation, with an operative mortality within three weeks of operation of less than 1.3%. This does not include 78 neck dissections.

## Classification

I have stated on previous occasions that for some time there has been considerable confusion regarding the classification of carcinoma of the larynx. It seems to me the time has come when a committee of laryngologists from the national societies and the College of Surgeons should be formed to definitely classify carcinoma as to its anatomical origin, and not as it is today. The old terms of intrinsic and extrinsic cancer should be abolished, for they are of no value in giving any information as to the site, prognosis, and treatment of these lesions. The following classification would be much more significant:

A. True cordal cancer, that which is confined to either vocal cord.

B. The anatomical site, which would designate the true origin of the growth, as in the epiglottis, ventricle, aryepiglottic fold, arytenoid, piriform sinus, and below the glottis.

These anatomical sites often involve problems in diagnosis and treatment. The laryngeal picture may be such that the lesion is so extensive that the original site of origin cannot be ascertained. However, if a careful history is taken, it will aid immeasurably in determining the original site.

It is true that at times the larynx is involved from a pharyngeal growth, but this is not primarily a laryngeal carcinoma.

In 1954 I presented before the American Academy of Ophthalmology and Otolaryngology a series of cases of carcinoma of the larynx based on the above classification. It showed that my best results following surgery were in the following order for five-year cures:

| | |
|---|---|
| Cords (70%) | 171 |
| Below the glottis (48%) | 78 |
| Ventricles (55%) | 54 |
| Epiglottis (65%) | 45 |
| Epiglottis and tongue (62%) | 23 |
| Aryepiglottic fold (16%) | 25 |
| Arytenoid (58%) | 25 |
| Piriform sinus (25%) | 20 |
| | 441 |

The results of the combined cases may be expressed in percentages as follows:

| | |
|---|---|
| Recurrence | approximately 20% |
| Successful cases | 70%—75% |
| Operative mortality | 1.3% |
| Deaths due to other causes than cancer | 10% |

Accepted for publication March 26, 1956.

Read before the Otolaryngological Section of the Sectional Meeting of the American College of Surgeons, Philadelphia, Feb. 15, 1956.

## Treatment

Thyrotomy, irradiation, and partial or total laryngectomy all have their proponents, and specific indications. These results can be improved through education to shorten the time between the onset of hoarseness and the inception of definite care if adequate examination is substituted for inapplicable therapeutic measures.

## Metastasis

Seventy-six cases out of the four hundred forty-one had cervical metastasis after total laryngectomy, for which a neck dissection was performed.

This brings up the subject of the evaluation of radical neck dissection at the time of the original operation or for cervical metastasis following laryngeal surgery. Palpable lymph nodes at the time of primary operation should include a neck dissection. It might be advisable to include a neck dissection on the side of the lesion without palpable lymph nodes at the time of the original operation in the subglottic and aryepiglottic regions, in the piriform sinus, ventricle, and large extensive lesions of the epiglottis on the lingual surface.

## Summary

The best time to treat extensive carcinoma of the larynx is at the first operation. The lymphatic drainage of these areas is such that metastasis takes place very early in the disease, and this area should be blocked off from the general system. The combined operation affords a better chance of cure than if we wait for clinical evidence of metastasis. These advantages should persuade the laryngologist to do this operation in selected cases. This operation is not indicated for true cordal lesions, and surgery is contraindicated in cases of massive firmly fixed lymph nodes.

The classification of laryngeal carcinoma should be standardized as to anatomical location.

The earlier the diagnosis is made the better the prognosis.

Complete removal of the primary growth is of utmost consideration in any operative procedure. The physical condition of the patient must be such that he is a reasonably good operative risk.

224 Delavan Ave. (4).

*Results in Seventy-Six Cases with Cervical Metastasis*

| Location of Lesion | Cases | Death Not Due to Cancer | Death Within 3 Weeks of Oper. | Years Lived Following Operation | | | | | Distant Metastasis |
|---|---|---|---|---|---|---|---|---|---|
| | | | | 1 | 2 | 3 | 4 | 5 | |
| Cords | 4 | 1 | 0 | 0 | 0 | 0 | 1 | 2 | 0 |
| Below glottis | 23 | 1 | 0 | 4 | 6 | 5 | 2 | 3 | 2 |
| Ventricle | 14 | 0 | 0 | 3 | 6 | 3 | 1 | 1 | 0 |
| Epiglottis | 8 | 1 | 0 | 1 | 0 | 2 | 2 | 1 | 1 |
| Aryepiglottic fold | 12 | 0 | 0 | 2 | 1 | 3 | 2 | 2 | 2 |
| Arytenoid | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 |
| Piriform sinus | 14 | 0 | 1 | 5 | 6 | 1 | 1 | 0 | 0 |

# Associated Laryngeal Paralysis

## Presentation of a Case of Bilateral Abductor Paralysis in a Patient with the Arnold-Chiari Deformity

ANTONIO RULLAN, M.D., M.Sc. (Med.), San Juan, Puerto Rico

By associated laryngeal paralysis is meant a paralysis of the vocal cord, which may be unilateral or bilateral, found in association with other neurologic signs due to pathology encroaching, usually, upon the last four cranial nerves.

This subject is of interest not only to the otolaryngologist, who is consulted because of symptoms of hoarseness or dysphagia, but also to the neurologist, internist, and chest surgeon, for we are not dealing with a disease entity but with signs of disease elsewhere, namely, within the cranium, nasopharynx, neck, or mediastinum.

According to Clerf, paralysis of the vocal cords is due in 90% of all the cases to peripheral lesions, i.e., all the types outside of the medulla. The other 10% of the cases are due to lesions in the medulla. Cody in 1946 reviewed 734 cases of all types of laryngeal paralysis and found that only 30 cases (i.e., 4% of the total) could be classified as associated laryngeal paralysis. Out of these 30 cases, he found that 21 cases were due to pathology at the medulla and 9 cases were due to pathology at the jugular foramen.

The clinical picture seen in associated laryngeal paralysis depends on two main factors: the basic pathology and the site of the lesion. The nerves usually involved have such a long and tortuous course that one readily understands why so many eponymic syndromes appeared in the literature from 1864 (Hughlings-Jackson) until 1917 (Villaret). H. Burger in 1934 summarized the entire subject and very wisely proposed a new classification as to the site where the pathology is located.

Clinically we can define associated laryngeal paralysis as follows:

A. Central lesions at the medulla (10% of all cases)
B. Peripheral lesions (90% of all cases)
   1. At the base of the skull
   2. At the lateral pharyngeal space
   3. At the infrahyoid part of the neck and mediastinum

Before proceeding with the clinical pictures, I should like to view some basic well-known anatomic facts:

1. The somatic motor component of the vagus nerve arises in the caudal part of the nucleus ambiguus located at the lateral part of the medulla. This area of the brain is supplied by the posterior-inferior cerebellar artery, which, in turn, is the largest branch of the vertebral artery.

2. The impulses brought to the cells of origin of the laryngeal nerve arise from both precentral regions of the cerebral cortex. This bilateral supranuclear innervation of the vocal cords was first demonstrated by Krause in 1884 and has been confirmed many times since.

3. The vagus nerve leaves the skull through the middle compartment of the jugular foramen included in the same sheath with the accessory nerve. The glossopharyngeal nerve also leaves the skull through the jugular foramen but in a separate sheath of dura mater lateral to and in front of the vagus and accessory nerves. The hypoglossal nerve leaves the skull through the anterior condylar foramen in the occipital bone.

Accepted for publication April 18, 1956.

From the School of Medicine, University of Puerto Rico.

Presented before the Fifth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, San Juan, Puerto Rico, April 12, 1956.

Other nerves involved in associated laryngeal paralysis are the sympathetic trunk, which lies medial to the vagus nerve in the neck and behind the common carotid artery, and the phrenic nerve, which arises from the cervical roots (mostly the fourth), descends across the front of the anterior scalenus muscle, and once within the thorax, crosses the root of the lung between the pericardium and the mediastinal pleura until it reaches the diaphragm, which it supplies.

The course of the vagus nerves deserves further explanation. The right vagus crosses in front of the subclavian artery, and the recurrent laryngeal branch hooks around the artery to reach the groove between the trachea and esophagus. The left vagus also descends in front of the subclavian artery; it enters the thorax behind the left innominate vein. As the vagus crosses over the arch of the aorta the recurrent laryngeal nerve arises on the left side of the arch and winds below the aorta at the point where the ligamentum arteriosum is attached and then ascends to the side of the trachea. Both recurrent laryngeal nerves ascend in the groove between the trachea and esophagus, pass under the lower border of the inferior pharyngeal constrictor muscle, and enter the larynx behind the articulation of the inferior cornu of the thyroid cartilage with the cricoid cartilage. It supplies the intrinsic muscles of the larynx except the cricothyroid muscle, which is supplied by the external branch of the superior laryngeal nerve, which arises from the vagus near the base of the skull. This muscle is the main tensor of the vocal cord.

Any pathology affecting the vagus nerve before the superior laryngeal nerve branches off will produce a complete paralysis of the true vocal cord. The cord will remain in the so-called cadaveric, or intermediate, position. The arytenoid cartilage will occupy a midposition and will be tilted forward. The vocal cord will be relaxed due to the lack of tensile stimulus normally received through the superior laryngeal nerve by the cricothyroid muscle. The cord is therefore flaccid and occupies a lower level than the unaffected cord. (In paralysis due to supranuclear pathology the cord is spastic.) The tip of the vocal process of the arytenoid cartilage is directed inward and appears as a definite prominence. There is a sufficiently wide glottis for respiration, but during phonation there is feebleness of voice due to air waste caused by inability of the normal cord to close the glottis.

When the recurrent laryngeal nerve alone is affected then an incomplete paralysis results. The vocal cord will remain at a median position; the tension of the cord will remain normal, since the cricothyroid muscle is still active. The voice will return to normal in 90% of the cases, but in the remaining 10% of the cases the cord will assume a cadaveric position due to atrophy resulting from disuse of the cricothyroid muscle. Paralysis of the left vocal cord is commoner than the right even if intrathoracic pathology is excluded. The ratio is 3:1.

4. The function of the other three cranial nerves usually involved is as follows: The glossopharyngeal nerve is mostly sensory in function and supplies the pharynx and posterior third of the tongue. Its only motor component supplies the stylopharyngeus muscle, which lifts the larynx and pharynx during phonation.

The accessory nerve is purely motor in function, and its cranial part supplies all the muscles of the soft palate except the tensor palati muscle, which is supplied by the mandibular branch of the trigeminal. Its spinal part supplies the sternocleidomastoid and trapezius muscles.

The hypoglossal nerve is also purely motor in function, and it supplies all the extrinsic muscles of the tongue except the palatoglossus, which is supplied by the cranial part of the accessory nerve.

Now that we have these basic anatomic facts refreshed in our minds, I should like to list the various associated laryngeal syndromes at the various levels where pathology is usually found:

## A. Laryngeal Syndromes at the Medulla

Ten per cent of all laryngeal syndromes are located at the medulla.

### 1. Syndromes Due to Vascular Lesions

(*a*) Thrombosis of the posterior-inferior cerebellar artery. The so-called Wallenberg syndrome is produced, consisting of ipsilateral paralysis of the soft palate, pharynx, and larynx, with dysphagia and dysphonia; ipsilateral anesthesia of the face for pain and temperature sensations, with loss of the corneal reflex; ipsilateral Horner's syndrome, and ipsilateral cerebellar asynergia and hypotonus. There is contralateral loss of pain and temperature sensations in the limbs and trunk. Sixth, seventh, and eighth nerve involvement on the side of the lesion has occasionally been reported.

(*b*) Lesions at the nucleus ambiguus and at the spinothalamic tract, as seen in thrombosis of the vertebral artery, will produce the syndrome of Avellis. There is ipsilateral paralysis of the soft palate, pharynx, and vocal cord, with contralateral loss of pain and temperature sensations in the trunk and extremities.

(*c*) Thrombosis of the vertebral artery below the point of origin of the posterior-inferior cerebellar artery will produce the snydrome of Cestan-Chenais. We find the same signs as in the Wallenberg syndrome plus contralateral hemiplegia.

(*d*) Cody and Furstenberg both believe that occasionally a case is seen due to vasoconstriction of the radicular arteries to the motor nuclei, producing a transient paralysis of the vocal cord, sometimes lasting only a few hours (seven hours in one case).

### 2. Syndromes Due to Intramedullary or Extramedullary Involvement

Syndromes of intramedullary or extramedullary involvement may be the result of vascular lesions (aneurysms, thromboses, hemorrhages) or may be found in association with syringobulbia, syringomyelia, multiple sclerosis, polioencephalitis, neoplasms, and trauma.

(*a*) Lesions at the entire nucleus ambiguus and the hypoglossus nucleus (10th, 11th, and 12th nerves), the so-called Jackson syndrome. There is ipsilateral flaccid paralysis of the soft palate, pharynx, and larynx, with flaccid weakness and atrophy of the sternocleidomastoid and trapezius muscles and of the tongue.

The vagoaccessory (10th and 11th nerves), or Schmidt, syndrome is due to a lesion at the nucleus ambiguus and of the bulbar and spinal nuclei of the accessory nerve. There is ipsilateral vocal cord paralysis, with flaccid weakness and atrophy of the sternocleidomastoid and trapezius muscles.

Tapia syndrome (10th and 12th nerves) is characterized by ipsilateral paralysis of the soft palate, pharynx, and vocal cord and paralysis and atrophy of the tongue.

## B. Laryngeal Syndromes of Peripheral Origin

### 1. Lesions at the Base of the Skull

(*a*) Tumors of the nasopharynx, chordomas, fractures, osteopetrosis, and aneurysm of the internal carotid artery will usually involve the jugular foramen and produce what is called Vernet's syndrome (9th, 10th, and 11th nerves). There is ipsilateral paralysis of the soft palate, pharynx, and vocal cord, ageusia of the posterior third of the tongue, and paralysis of the shoulder girdle.

(*b*) Neuritis due to toxic factors, avitaminosis, or virus infection producing the jugular foramen syndrome have been reported by Suehs, Alcantara and de Ocampo, Clerf, and Engström and Wohlfart.

Font in 1952 reported two cases due to virus infection (one of which was under my care). Both cases completely recovered within six months after the syndrome appeared.

(*c*) Platybasia producing the Arnold-Chiari deformity, which consists of displacement of the cerebellar tissue into the medulla or the spinal cord. The neurologic picture consists of bulbar and cerebellar

signs, with palsies of the 9th, 10th, 11th, and 12th cranial nerves, involvement of the pyramidal tracts and sensory pathways, and cerebellar manifestations.

I should like to report what is probably the first case in the literature of bilateral abductor paralysis of the vocal cord in a patient with the Arnold-Chiari deformity.

A 48-year-old white woman was admitted to the San Juan City Hospital on Jan. 25, 1955, complaining of dysphagia, hoarseness, dyspnea, visual disturbances, and ataxia since May, 1954.

The positive neurological findings were reported as follows: Vertical type of nystagmus to the left and a horizontal type of nystagmus to the right. The deep tendon reflexes were very hyperactive, and the lower abdominal reflexes were absent. Special cerebellar tests were negative. A positive Romberg test was found. A tentative diagnosis of multiple sclerosis was made. The rest of the physical examination was normal except for a labile blood pressure and the laryngeal examination. Both true vocal cords were paralyzed in the midline in the so-called cadaveric position with flaccidity of the cords and the arytenoid cartilages tilted forward. The glottic chink was no more than 2 mm. Phonation was clear. A tracheotomy was done and the patient relieved of her dyspnea. Nevertheless, postoperatively she had two episodes of respiratory and cardiovascular distress which could not be explained. Her tracheotomy cannula was always clean and patent. Patient was discharged on Feb. 21, 1955, to the Ear, Nose and Throat Clinic and also to the Neurology Clinic, since her diagnosis was not yet too clear.

Neurologic examination repeated on Oct. 27, 1955, revealed the same clinical picture, but this time the neurologist thought of platybasia. X-ray films of the skull revealed "protrusion of the odontoid process of the first cervical vertebra into the foramen magnum indicating platybasia." A myelogram done Feb. 18, 1956, revealed "partial obstruction at the level of the axis with a smooth concave deformity of the radio-opaque column. The findings are compatible with an obstructive lesion at the region such as seen in Arnold-Chiari syndrome or by an intradural extramedullary tumor."

On Feb. 20, 1956, the neurosurgeon did a craniotomy and a laminectomy of the first and second cervical vertebrae. His findings were as follows: "The cerebellar tonsils descended below the inferior border of the atlas completely surrounding the posterior half of the superior segment of the spinal cord. Both tonsils were closely adhered at the midline." This fits the findings described by Arnold and later by Chiari.

The patient had an uneventful course and was discharged March 11, 1956. My last laryngeal examination, done April 5, 1956, revealed no change at all in the position of the vocal cords. The patient is wholly dependent on her tracheotomy tube. There has been no change, either, in the original neurologic findings.

### 2. Lesions at the Lateral Pharyngeal Space

Pathology at the lateral pharyngeal space, such as tumors or infections, may involve the cranial nerves (9th, 10th, 11th, and 12th) and also the cervical sympathetic fibers. The syndrome of Villaret is produced. There is ipsilateral paralysis of the soft palate, pharynx, and vocal cord, atrophy of the tongue and ageusia of its posterior third, as well as Horner's syndrome (enophthalmos, ptosis, and miosis).

When the pathology at this space does not involve the sympathetic fibers then we have the Collet-Sicard syndrome.

Fig. 1.—Chest film of a man who died of a superior mediastinum malignancy. The left vocal cord and the left leaf of the diaphragm were paralyzed: Klinkert syndrome.

### 3. Lesions at the Infrahyoid Part of Neck and Mediastinum

Mediastinal lesions and those at the infrahyoid part of the neck will produce associated laryngeal paralysis only when they involve the vagus nerve and either the spinal

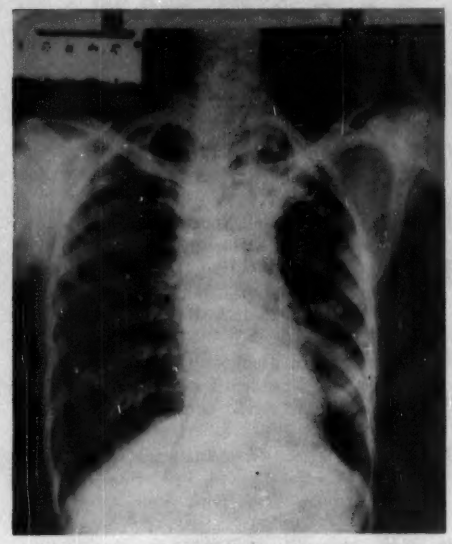

Fig. 2.—Film showing moderate retraction of the left lung, with fibrocalcific disease of the upper lobe and probable cavitation of the apex by tuberculosis. There is also diffuse infiltration of the left lower lung. The left vocal cord is paralyzed in the median position.

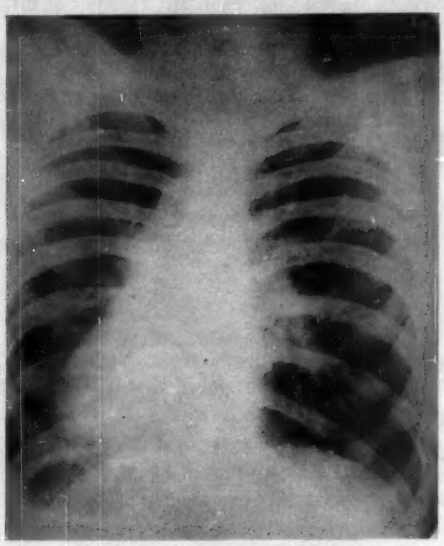

Fig. 4.—Case of interauricular septal defect with cardiac enlargement. Laryngoscopy revealed the left true vocal cord to be paralyzed in the cadaveric position. This case falls into the 10% with recurrent laryngeal nerve paralysis that in time undergoes complete paralysis due to atrophy resulting from disuse of the cricothyroid muscle.

part of the accessory nerve. (Gigonoux

Fig. 3.—Esophagram of a man with a carcinoma of the cricopharyngeal area. The right vocal cord is paralyzed in the median position.

snydrome) or the phrenic nerve (Klinkert syndrome). Most of the etiologic factors found here usually will affect only the recurrent laryngeal nerve and thus do not fall into the category of associated laryngeal paralysis.

They are listed here as an aid in differential diagnosis:

(*a*) Mechanical factors
1. Aneurysm of the aorta
2. Cardiac enlargement due to various causes, such as congenital heart disease, rheumatic fever with mitral stenosis and a dilated left auricle
3. Pericarditis due to various causes
4. Diverticulum of the hypopharynx

(*b*) Inflammatory factors
1. Tuberculous fibrosis of the lung apex
2. Tuberculous adenitis of the neck

(*c*) Tumors (main cause of unilateral laryngeal paralysis)
1. Carcinoma of the esophagus (commonest cause in this group)
2. Carcinoma of the lung
3. Mediastinal tumors, either primary or metastatic

4. Benign and malignant tumors of the thyroid gland
5. Tumors of the carotid body and of the trachea

(*d*) Trauma

1. Surgical: main cause of bilateral laryngeal paralysis (usually seen after thyroidectomies or esophageal surgery)
2. Nonsurgical: such trauma to the neck as fractures of the clavicle, stab and gunshot wounds, and birth injury

Figures 1 through 4 illustrate intrathoracic pathology producing vocal cord paralysis.


## Summary

The subject of associated laryngeal paralysis is completely reviewed, and the different syndromes are classified as to the site where the pathology is usually located, namely: in the medulla, base of the skull, neck, and mediastinum. A case of bilateral abductor laryngeal paralysis in a patient with the Arnold-Chiari deformity is reported.



Dr. Ricardo Cordero, a neurosurgeon, handled this case.

Ponce de Leon Ave. 1803.


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# News and Comment

### PERSONAL NEWS

**Albert P. Seltzer.**—Albert P. Seltzer, M. D., has been appointed Chief of the Department of Otolaryngology at Philadelphia General Hospital, Blockley Division.

## Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

# An Experimental Study of Audiometry in Simulated Partial Deafness

LAWRENCE K. GUNDRUM, M.D., Los Angeles

Many physicians, including otologists, are of the opinion that audiometric tests, repeated at various time intervals, constitute a reliable basis on which to detect or reject malingering, for it would be extremely difficult for an ordinary person to repeat a pure-tone threshold were he, or she, to feign partial deafness. According to Reger,* it is conceivable that a musician whose ears are attuned to fine modulations of sound, or a specialist in the physics of sound, might successfully simulate a partial loss of hearing, but it is improbable that the average person could achieve such a simulation.

In order to ascertain the correctness of this viewpoint the study reported here was made with the assistance of two groups, namely:

1. A selected, highly cooperative, voluntary group of 50 graduate and student nurses, 18 to 25 years old, attached to the Queen of Angels' Clinic. All were given a preliminary hearing test and found to be possessed of normal hearing. They were then instructed to make every effort to simulate partial deafness, and, especially, to remember the pure-tone threshold at which they had elected to declare a loss of hearing. Because of their educational background all grasped the reason for the experiment readily and submitted themselves most intelligently to the tests.†

Accepted for publication April 2, 1956.

From the Department of Otolaryngology, Queen of Angels' Clinic.

*Reger, S.: Personal communication to the author.

†Lucille Graves, R. N. and audiologist, assisted in giving the tests to this group.

TABLE 1.—*Results of Hearing Tests in Group 1 (Simulated Deafness Cases)*

| Case | First Audiogram Date | First Audiogram Loss A.M.A. % L | First Audiogram Loss A.M.A. % R | Second Audiogram Date | Second Audiogram Loss A.M.A. % L | Second Audiogram Loss A.M.A. % R | Variation Between 1st and 2nd Audiograms A.M.A. % L | Variation Between 1st and 2nd Audiograms A.M.A. % R |
|---|---|---|---|---|---|---|---|---|
| 1 | 5/15/52 | 50 | 98 | 6/ 2/52 | 70 | 98 | 20+ | 0 |
| 2 | 5/15/52 | 53 | 43 | 6/ 2/52 | 73 | 63 | 20+ | 20+ |
| 3 | 7/31/52 | 73 | 75 | 8/ 2/52 | 78 | 66 | 5+ | 9− |
| 4 | 7/31/52 | 46 | 84 | 8/ 2/52 | 39 | 64 | 7− | 20− |
| 5 | 7/31/52 | 5 | 11 | 8/13/52 | 12 | 24 | 7+ | 13+ |
| 6 | 3/18/53 | 51 | 29 | 4/ 9/53 | 48 | 69 | 3− | 40+ |
| 7 | 3/18/53 | 14 | 7 | 4/ 7/53 | 1 | 22 | 13− | 15+ |
| 8 | 3/18/53 | 34 | 52 | 4/ 7/53 | 55 | 62 | 21+ | 10+ |
| 9 | 3/19/53 | 85 | 58 | 4/ 7/53 | 33 | 12 | 52− | 46− |
| 10 | 3/19/53 | 82 | 78 | 4/ 7/53 | 91 | 82 | 9+ | 4+ |
| 11 | 4/ 7/53 | 92 | 94 | 4/14/53 | 100 | 99 | 8+ | 5+ |
| 12 | 4/ 7/53 | 92 | 63 | 4/14/53 | 71 | 70 | 21− | 7+ |
| 13 | 3/18/53 | 85 | 98 | 4/ 7/53 | 95 | 98 | 10+ | 0 |
| 14 | 4/ 9/53 | 55 | 63 | 5/ 5/53 | 65 | 52 | 10+ | 11− |
| 15 | 4/10/53 | 40 | 49 | 4/30/53 | 50 | 60 | 10+ | 11+ |
| 16 | 4/10/53 | 44 | 45 | 5/ 3/53 | 58 | 64 | 14+ | 19+ |
| 17 | 5/ 5/53 | 24 | 27 | 5/13/53 | 41 | 50 | 17+ | 23+ |
| 18 | 4/ 9/53 | 37 | 33 | 4/23/53 | 67 | 59 | 30+ | 26+ |
| 19 | 2/18/54 | 14 | 11 | 3/ 7/54 | 12 | 8 | 2− | 3− |
| 20 | 2/24/54 | 10 | 5 | 3/15/54 | 22 | 22 | 12+ | 17+ |
| 21 | 5/ 3/55 | 18 | 10 | 5/23/55 | 5 | 5 | 13− | 5− |
| 22 | 4/15/54 | 100 | 100 | 5/ 4/54 | 25 | 27 | 75− | 73− |
| 23 | 5/ 5/55 | 9 | 20 | 5/13/55 | 44 | 34 | 35+ | 14+ |
| 24 | 5/ 5/55 | 14 | 5 | 5/27/55 | 19 | 25 | 5+ | 20+ |
| 25 | 4/21/54 | 18 | 72 | 5/ 6/54 | 54 | 40 | 36+ | 32− |
| 26 | 4/21/54 | 1 | 17 | 5/10/54 | 18 | 12 | 17+ | 5− |
| 27 | 4/ 6/53 | 27 | 5 | 4/28/53 | 38 | 41 | 11+ | 36+ |
| 28 | 7/ 9/53 | 10 | 5 | 7/28/53 | 30 | 40 | 20+ | 35+ |
| 29 | 6/ 1/53 | 89 | 100 | 6/20/53 | 62 | 70 | 27− | 30− |
| 30 | 7/ 1/54 | 90 | 84 | 7/18/54 | 81 | 79 | 9− | 5− |
| 31 | 9/ 4/53 | 89 | 77 | 9/20/54 | 58 | 27 | 31− | 50− |
| 32 | 10/1/53 | 50 | 12 | 10/15/53 | 40 | 38 | 10− | 26+ |
| 33 | 4/21/54 | 13 | 35 | 4/30/54 | 1 | 5 | 12− | 30− |
| 34 | 4/21/54 | 15 | 6 | 4/30/54 | 25 | 15 | 10+ | 9+ |
| 35 | 4/28/54 | 35 | 37 | 4/21/55 | 20 | 18 | 15− | 19− |
| 36 | 4/21/54 | 8 | 5 | 4/30/54 | 14 | 14 | 6+ | 9+ |
| 37 | 5/ 1/54 | 10 | 10 | 4/21/55 | 7 | 8 | 3− | 2− |
| 38 | 4/20/55 | 5 | 20 | 4/21/55 | 10 | 10 | 5+ | 10− |
| 39 | 4/10/54 | 18 | 30 | 4/20/54 | 18 | 38 | 0 | 8+ |
| 40 | 4/22/54 | 65 | 63 | 5/ 6/55 | 42 | 21 | 23− | 42− |
| 41 | 4/22/54 | 67 | 56 | 5/ 6/54 | 78 | 65 | 11+ | 9+ |
| 42 | 4/22/54 | 43 | 33 | 5/ 6/54 | 23 | 23 | 20− | 10− |
| 43 | 4/22/54 | 50 | 21 | 5/ 5/54 | 53 | 34 | 3+ | 13+ |
| 44 | 5/ 6/54 | 13 | 7 | 5/15/54 | 28 | 19 | 15+ | 12+ |
| 45 | 10/27/54 | 35 | 28 | 5/ 5/55 | 21 | 14 | 14− | 14− |
| 46 | 4/20/54 | 13 | 20 | 4/30/54 | 38 | 43 | 25+ | 23+ |
| 47 | 5/ 5/54 | 32 | 42 | 5/11/55 | 37 | 31 | 5+ | 11− |
| 48 | 4/20/54 | 7 | 4 | 5/ 5/54 | 1 | 3 | 6− | 1− |
| 49 | 4/20/54 | 38 | 30 | 5/10/55 | 10 | 12 | 28− | 18− |
| 50 | 4/20/54 | 11 | 11 | 4/20/55 | 22 | 5 | 11+ | 6− |

A plus sign indicates the second audiogram was higher; a minus sign, the second audiogram was lower.

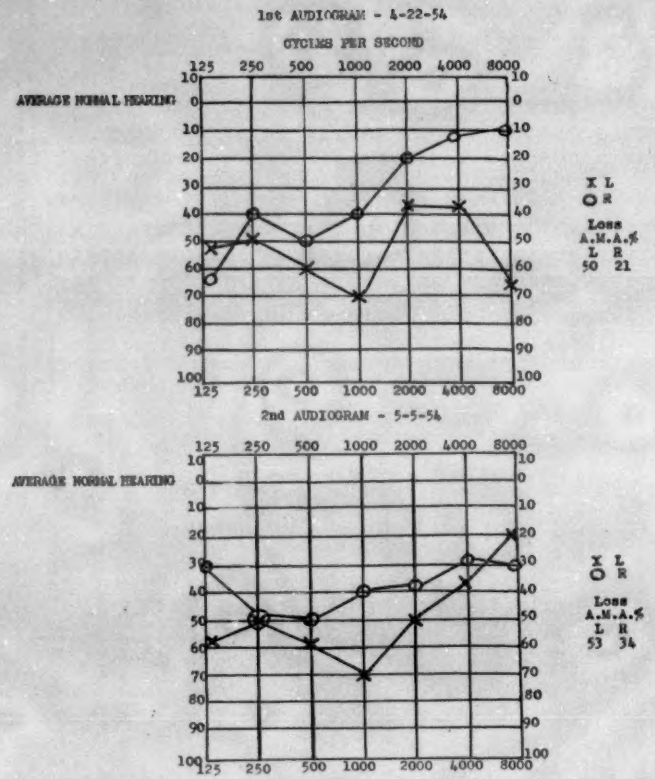


Fig. 1 (Case 43).—Audiograms typical of those found in Group 1 (simulated deafness cases).

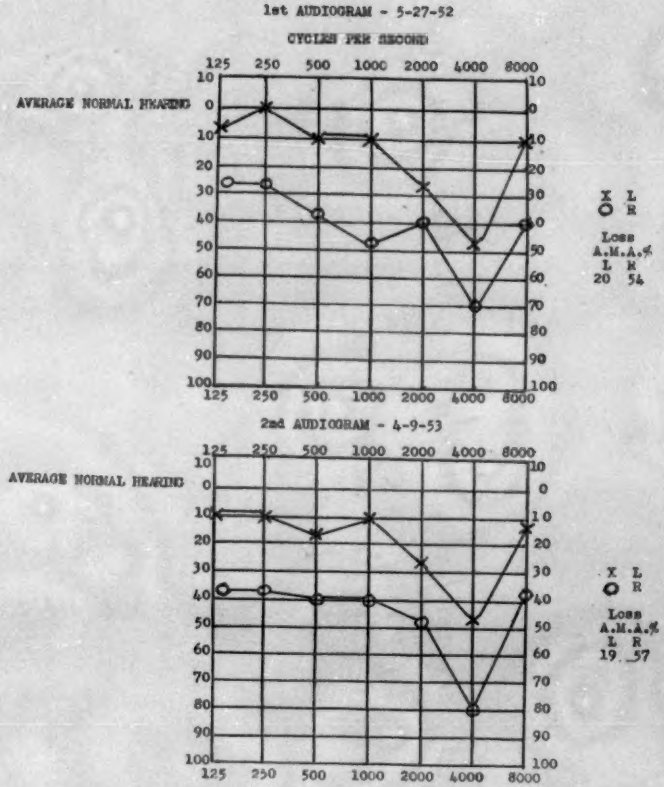


Fig. 2. (Case 7).—Audiograms typical of those found in Group 2 (accident cases).

TABLE 2.—*Results of Hearing Tests in Group 2 (Accident Cases)*

| Case | First Audiogram Date | First Audiogram Loss A.M.A. % L | First Audiogram Loss A.M.A. % R | Second Audiogram Date | Second Audiogram Loss A.M.A. % L | Second Audiogram Loss A.M.A. % R | Variation Between 1st and 2nd Audiograms A.M.A. % L | Variation Between 1st and 2nd Audiograms A.M.A. % R |
|---|---|---|---|---|---|---|---|---|
| 1 | 10/31/51 | 32 | 100 | 11/ 5/51 | 36 | 100 | 4+ | None |
| 2 | 11/13/50 | 11 | 15 | 11/17/50 | 10 | 15 | 1− | None |
| 3 | 3/30/53 | 77 | 0 | 4/ 2/53 | 77 | 0 | None | None |
| 4 | 7/15/54 | 95 | 81 | 7/19/54 | 94 | 89 | 1− | 8+ |
| 5 | 10/18/49 | 84 | 80 | 10/21/49 | 87 | 83 | 3+ | 3+ |
| 6 | 7/11/52 | 1 | 5 | 1/ 4/53 | 1 | 6 | None | 1+ |
| 7 | 5/27/52 | 20 | 54 | 4/ 9/53 | 19 | 57 | 1− | 3+ |
| 8 | 3/15/52 | 7 | 82 | 2/27/53 | 4 | 83 | 3− | 1+ |
| 9 | 3/11/55 | 23 | 16 | 3/19/55 | 20 | 19 | 3− | 3+ |
| 10 | 12/28/50 | 70 | 77 | 1/ 9/51 | 70 | 76 | None | 1− |
| 11 | 2/12/54 | 6 | 3 | 4/20/54 | 6 | 7 | None | 4+ |
| 12 | 1/17/55 | 51 | 55 | 1/24/55 | 59 | 58 | 8+ | 3+ |
| 13 | 1/13/53 | 98 | 25 | 1/23/53 | 100 | 28 | 2+ | 3+ |
| 14 | 6/27/49 | 100 | 99 | 6/28/49 | 98 | 99 | 2− | None |
| 15 | 2/ 6/53 | 3 | 44 | 2/ 9/53 | 8 | 41 | 5+ | 3− |
| 16 | 1/ 7/55 | 28 | 44 | 1/15/55 | 25 | 52 | 3− | 8+ |
| 17 | 1/18/54 | 70 | 6 | 9/ 9/54 | 60 | 2 | 10− | 4− |
| 18 | 10/ 1/51 | 10 | 9 | 10/22/51 | 12 | 7 | 2+ | 2− |
| 19 | 5/27/55 | 45 | 0 | 6/ 2/55 | 46 | 0 | 1+ | None |
| 20 | 2/29/52 | 67 | 44 | 3/ 4/52 | 62 | 46 | 5− | 2+ |
| 21 | 1/24/52 | 94 | 98 | 1/31/52 | 96 | 99 | 2+ | 1+ |
| 22 | 8/31/53 | 31 | 6 | 9/ 4/53 | 31 | 6 | None | None |
| 23 | 2/ 6/53 | 0 | 5 | 2/12/53 | 0 | 5 | None | None |
| 24 | 11/18/54 | 12 | 18 | 11/19/54 | 7 | 13 | 5− | 5− |
| 25 | 4/18/53 | 40 | 32 | 5/20/53 | 38 | 37 | 2− | 5+ |
| 26 | 1/22/52 | 16 | 94 | 1/23/52 | 18 | 96 | 2+ | 2− |
| 27 | 6/26/52 | 50 | 4 | 6/27/52 | 47 | 4 | 3− | None |
| 28 | 4/ 5/54 | 9 | 9 | 1/24/55 | 11 | 13 | 2+ | 4+ |
| 29 | 6/26/52 | 91 | 86 | 1/ 5/53 | 94 | 84 | 3+ | 2− |
| 30 | 3/21/55 | 11 | 13 | 3/22/55 | 11 | 12 | None | 1− |
| 31 | 7/18/52 | 0 | 29 | 7/29/52 | 0 | 35 | None | 6+ |
| 32 | 5/17/51 | 20 | 3 | 9/ 4/51 | 21 | 3 | 1+ | None |
| 33 | 6/ 9/55 | 23 | 77 | 9/15/55 | 21 | 68 | 2− | 9− |
| 34 | 2/25/54 | 13 | 7 | 8/ 9/54 | 14 | 7 | 1+ | None |
| 35 | 8/12/54 | 1 | 9 | 8/13/54 | 2 | 7 | 1+ | 2− |
| 36 | 9/16/52 | 8 | 6 | 9/18/52 | 9 | 10 | 1+ | 4+ |
| 37 | 9/26/52 | 14 | 26 | 9/29/52 | 19 | 21 | 5+ | 5− |
| 38 | 11/18/54 | 70 | 84 | 12/30/54 | 71 | 85 | 1+ | 1+ |
| 39 | 11/24/53 | 5 | 4 | 12/ 8/53 | 5 | 3 | None | 1− |
| 40 | 4/14/53 | 10 | 14 | 6/ 3/54 | 8 | 13 | 2− | 1− |
| 41 | 1/17/55 | 7 | 49 | 1/20/55 | 4 | 47 | 3− | 2− |
| 42 | 8/ 5/52 | 16 | 23 | 2/12/53 | 15 | 26 | 1− | 3+ |
| 43 | 12/15/50 | 89 | 9 | 12/29/50 | 91 | 16 | 2+ | 7+ |
| 44 | 9/15/53 | 2 | 11 | 9/22/53 | 3 | 15 | 1+ | 4+ |
| 45 | 5/17/55 | 4 | 34 | 5/20/55 | 3 | 35 | 1− | 1+ |
| 46 | 4/27/54 | 11 | 10 | 8/ 6/54 | 12 | 14 | 1+ | 4+ |
| 47 | 1/22/53 | 5 | 13 | 1/23/53 | 3 | 12 | 2− | 1− |
| 48 | 11/11/54 | 38 | 72 | 11/13/54 | 36 | 78 | 2− | 6+ |
| 49 | 9/15/50 | 17 | 7.5 | 10/30/51 | 11 | 7 | 6− | 0.5− |
| 50 | 10/ 8/53 | 96 | 92 | 10/22/53 | 95 | 84 | 1− | 8− |

A plus sign indicates the second audiogram was higher; a minus sign, the second audiogram was lower.

2. A group of 50 patients, accident cases treated in my office, who had all suffered head injuries resulting in actual partial deafness. They were not informed that they were being tested for the purpose of comparison. Some were given the repeat test a few days after the first; others, at longer intervals extending to one year and over.

All tests were made, and checked, by trained and skilled technicians.

No similar study, involving normal subjects, could be found in the literature.

Tables 1 and 2 show the results of the tests.

The figures appearing in the variation columns speak for themselves. It is obvious that in the first group, with the exception of Cases 19 and 37, the subjects were unable to repeat a tone threshold approaching the first. These two cases represent nurses who were known to have been musicians before engaging in the nursing profession.

On the other hand, the tests of the second group reveal relatively small variations between the two audiograms.

Not only in terms of percentages did the findings between the two groups differ sensibly but also in the tone curves, which, for obvious reasons, cannot be reproduced in toto here. The diagrams in Figures 1 and 2 are good examples of both groups. It will be noted that Figure 2 shows the characteristic dip at 4000 cps, which is absent in Figure 1 as well as in all the other audiograms pertaining to Group 1.

## Comment

Although audiometry cannot be called infallible, I believe, judging from the experiment reported here, that it, nevertheless, is a reliable method by which to determine actual or simulated partial loss of hearing. I may add that it has the advantage of being a relatively simple procedure, easily carried out in an office.

## Summary

The results of a comparative study of audiometry in simulated and actual partial deafness are reported.

The repeated audiograms obtained from a group of 50 normal graduate and student nurses, instructed to feign partial loss of hearing, are compared with those from 50 patients, accident cases, showing actual partial deafness.

No similar study can be found in the literature.

The variations between the first and second audiograms are relatively great in the first group and small in the second.

The diagrams illustrate the difference in the character of the curves.

# Continuous, or Sweep-Frequency, Audiometry

## Its Technique in Adults and Children

H. A. E. van DISHOECK, M.D., Leyden, Netherlands

### Introduction

Pure-tone threshold audiometry provides us with a restricted knowledge about the hearing function and, especially, about the hearing of normal speech. However, the indispensable information on the hearing capacity for different frequencies and thus on the condition of every part of the basilar membrane can only be obtained by pure-tone audiometry. Here, too, the speech range will be part of our main concern, and for this reason our method of measuring should be as accurate as possible in regard to both intensity and the frequencies involved. The same holds true for the higher frequencies when we wish to detect an impending noise trauma.

For such demands octave audiometry is an imperfect means, as with this technique a very restricted number of frequencies are tested and the parts of the basilar membrane lying between these frequencies are neglected. Thus only a rough idea of the hearing abilities is obtained. However, the ideal of pure-tone threshold audiometry cannot be other than the exact recording of the hearing loss for all tones within the audible range. It is astonishing that so far most otologists have been content with establishing the hearing acuity of their patients for only a few points in the tonal scale. This is as surprising as if we observed a piano tuner checking the tuning of a piano for the octaves of C only. This strange behavior is due to a confirmed habit of thought brought about by the tyranny of the tuning forks at the time of the development of audiometry, and it has persisted down to our own time.

Continuous audiometry aims at our ideal, namely, the recording of the hearing acuity for all frequencies, thus establishing the exact pattern of the auditory threshold. This is realized by using a sweep-frequency audiometer and by presenting to the patient the complete frequency range from 16 to 16000 cps on a fixed intensity level. This is the opposite of the procedure used in octave audiometry, in which the frequency is fixed and the intensity varied.

### The Continuous Audiometer

For continuous audiometry a sweep-frequency audiometer is essential. Not every sweep-frequency audiometer, however, is fit for use. The main requirement is that the frequency characteristic of the headphone be straight, in order to obtain as the average normal zero isophone a uniformly curved line without peaks and dips. Some audiometers are delivered with a satisfactory headphone, whereas for others it is necessary to order a more expensive headphone.

Starting in 1942, my co-workers and I adapted a Western Electric audiometer to this purpose and, later, a Peters audiometer. At present we use the specially built continuous audiometer of Peekel. In this last audiometer the differences of the sensibility of the normal ear are compensated so that the zero isophone of a normal patient is straight from 200 to 8000 cps. The frequency of 1000 cps is in the middle of the

Accepted for publication March 27, 1956.

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approximately logarithmic scale and 150 degrees to the left is occupied by the frequencies from 80-1000 cps and 150 degrees to the right by frequencies from 1000-12000 cps.

In the Peekel audiometer the frequency is within 3% of the value indicated on the audiogram chart. As beat-frequency oscillators are known to drift considerably in frequency, in this audiometer an R-C generator is used. As it is not practically possible to cover the whole desired frequency range by one rotation of the tuning condenser, an overlapping switch-over at 1000 cps proved to be unavoidable. Thus a very stable instrument is constructed that needs no frequency calibration prior to use. This arrangement is highly suitable for the sweep-frequency technique. One can easily determine the thresholds at different intensities of the high frequencies after having obtained these values for the lower frequencies. Thus only one switch-over is needed. The attenuator is adjustable in steps of 5 db. with a precision lower than +1 db. to —1 db.

### The Audiocard

As continuous audiometry deals with the determination of the perceptible frequency span on different intensity levels, it is possible to note the results in frequency ranges and afterward to draw a hearing-loss audiogram from these data. For this purpose Veckmans (1949) proposed a practical method.

Time is saved by using a direct registration on a specially arranged audiocard. The audiocard is fixed on a supporting table on the audiometer. A shaft which is fixed on the frequency dial passes through an opening in the supporting table and the audiocard. An arm is attached perpendicularly to the end of this shaft. When the frequency dial is turned the arm passes over the audiocard so that the beginning and ending of the patient's hearing at a certain intensity level can be marked directly on the card. For this purpose there is an intensity calibration on the arm, 5 mm. for 10 db. These circles on the audiocard represent intensity levels. A zero isophone established by the factory is noted on the audiocard.

The introduction of a chart fixed on the audiometer with an adjustable zero line has a big advantage in connection with the threshold calibration of the instrument. By taking a sufficient number of audiograms for normal ears, in the same environment where the otologist will use his audiometer, the zero line can be drawn on the chart. As many audiometers are used in conditions that differ considerably from those in the factory, a factory-provided zero line often needs correction, especially in the low frequencies. Also, in octave audiometry the zero setting depends on the noise conditions in which the factory has calibrated the audiometer, and it is often not easy to adapt an octave audiometer to other noise conditions. In general the room noise is most annoying for making audiograms of almost normal ears. As deafness increases the influence of the room noise on the audiogram diminishes. Repeated control of the audiometer is possible by checking the zero isophone on the card from time to time with the mean threshold curve of normal young subjects. (A great advantage is that the audiogram, when compared with the normal zero isophone, is at once ready as a hearing-loss curve. Thus much time is saved, for the drawing of a curve afterward is superfluous.)

Some otologists do not like the curved audiogram obtained in this way and prefer a straight hearing-loss audiogram. This can be achieved by using a simple device like the wave scale of an ordinary radio. We personally prefer the polar inscription because of its long tonal scale on a relatively small card.

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In order to make a continuous audiogram the patient is instructed to indicate in some way the moment he begins to hear and the moment he ceases to hear the sweep tone.

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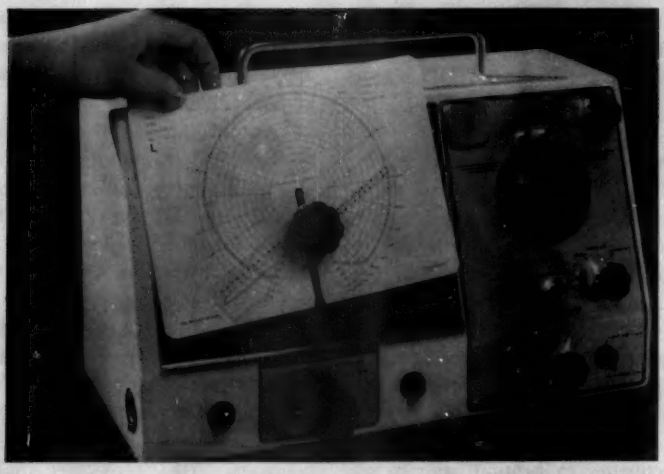

Fig. 1.—Peekel audiometer: Polar inscription. The zero isophone is empirically determined in young adults in a relatively silent room. It is a circle segment straight from 200-8000 cps.

This may be done by speaking, by raising or lowering a finger, or by switching a light on and off.

In the course of time we adopted certain rules for the recording of a continuous audiogram. An example of the different operations, each following from the result of the former one, is given in Figure 2.

1. Begin on a clearly audible intensity level. If on this level a reduced perceptible

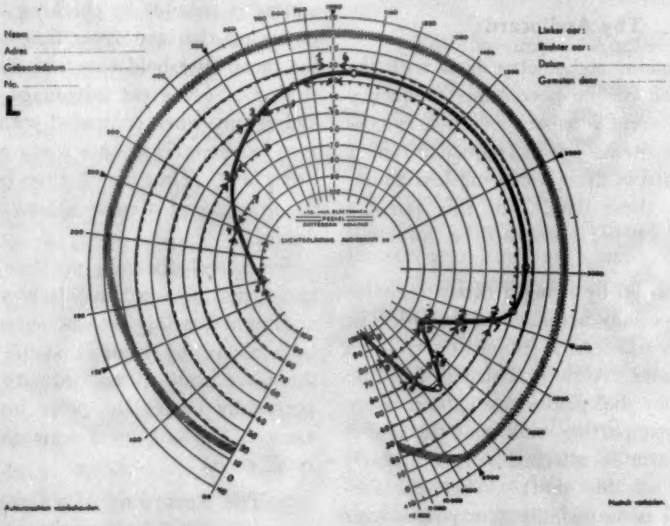


Fig. 2.—Recording of a continuous audiogram: The double row of x's represents the empirical normal zero isophone. The numbers indicate the sequence of the measurements and the arrows the direction from which the threshold is looked for. The broken line indicates no hearing; the dotted line, hearing.

frequency span is found, we know that on lower intensity levels hearing must be looked for within the range and not outside of it. This knowledge saves time.

2. Avoid loud tones. An effective procedure is to move the frequency dial back and forth with diminishing amplitude across the threshold area until the patient's indications are limited to a very small area. Start with determination of the thresholds of the low tones of different intensities. Then, after switching over, the same is done for the high frequencies.

3. Establish the main parts of the audiogram with 10 db. steps, the threshold in the critical speech area and the bottom of a dip or the top of a hearing isle with 5 db. steps, and the less interesting parts of the audiogram with 20 db. steps (Fig. 2).

### Masking of the Ear Not Under Investigation

Every masking method has to aim at the fulfilment of two basic requirements: 1. The test tone must not be overheard by the ear not under investigation. 2. The masking noise must not be overheard by the ear under investigation to such a degree that the threshold of that ear is altered. In this respect we must rely on the isolating capacity of the head to prevent the signal and the noise from reaching the other ear. In air conduction this isolating effect on a signal measured on a number of one-sided deaf patients proved to be 30-70 db. In bone conduction, however, this value proved to be only 0-25 db. Thus the main difficulties are to be expected in bone-conduction measuring.

In using a masking noise of fixed intensity, as is mostly done, the two basic requirements will not be satisfied. Masking noises of more than 55 db. will reach the ear under investigation (across or around the head) with an intensity which may be disturbing. This noise of 55 db. will mask in the same ear a test tone of 40 db. Such an intensity may reach this ear from the other side in air conduction when the signal is 70 db. However, in bone conduction this may occur already when the signal is 40 db. On higher tone intensity levels than these values one therefore risks overhearing of the test tone because of insufficient masking, and when increasing the fixed masking noise intensity, one risks a threshold shift in the ear under investigation.

The only way out of these dilemmas is not to use a masking noise of fixed intensity, but to adjust masking noise and tone intensity to each other. As in continuous audiometry, we measure the width of the perceptible frequency span at a fixed intensity level by determining the thresholds at both sides; it is easy to choose for each intensity level the adjusted noise intensity.

Thus in air-conduction audiometry the ear not under investigation should be masked with a minimum noise intensity of 15 db. below the tone intensity level. It is also permitted to use a masking noise at the signal level. In bone-conduction audiometry the minimum noise intensity amounts to 15 db. above the tone intensity level, but we prefer to use the permitted noise intensity of 30 db. above the signal. In those cases in which there is a normal correlation between the bone- and air-conduction thresholds of the ear not under investigation these simple masking rules will do. If such a normal correlation does not exist because of conductive deafness in the ear not under investigation, additional masking above those minimum values is necessary in bone-conduction measurements. The additional noise intensity needed is equal to the rise of the air-conduction noise threshold as compared with the bone-conduction noise threshold. The noise intensity, however, should in general not exceed 55 db. above the tone intensity level. This corresponds to a difference between the bone- and air-conduction noise thresholds of 40 db. Cases in which this difference is higher are rare. In these cases one has to investigate individually whether it is allowable to surpass the maximum value of 55 db. above the tone intensity level. The lateralization of

the signal by the patient—the Weber test—is in some exceptional cases the only reliable indication (Burgemeestre, 1953).

### The Exactness of Continuous Audiometry as Compared with That of Octave Audiometry

Since in continuous audiometry the hearing acuity of all frequencies is determined, a sometimes astonishing abundance of details is found as compared with those in octave audiometry. This appears in the comparison of the continuous and the octave audiogram of the same patient (Fig. 3).

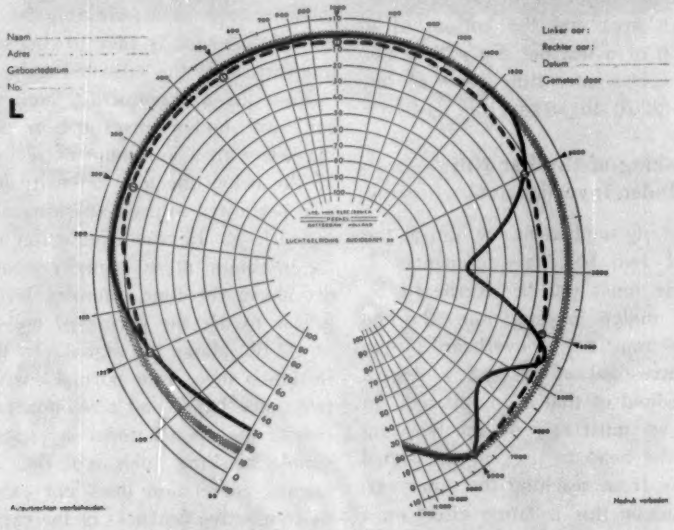


Fig 3.—Continuous audiogram compared with the octave audiogram of the same patient. The two dips appear in the continuous audiogram but not in the octave audiogram. The solid line represents the continuous audiogram; the broken line, the octave audiogram.

Thus continuous audiometry provides us with a much more exact knowledge of the function of the ear.

A question apart is the exactness of each determination in the two techniques; viz., to what degree do audiograms of the same patient repeated within a short time differ?

In order to study this highly important problem a continuous and an octave audiogram were made alternately of the same subject, in total six (v.d. Waal, 1954). The results in normal and impaired ears are reported in Tables 1 and 2.

From these investigations it appears that the average error in continuous audiometry is much smaller than that in octave audiometry. In impaired ears in both methods this error is greater, but the probability of a considerable error is much smaller in continuous than in octave audiometry. Moreover, the question was studied whether perhaps in perceptive deafness the error is greater than in conductive deafness (Table 3). This was calculated for 16 frequencies taken from the continuous audiograms of selected patients. No difference of any importance was found.

### Clinical Advantages of Continuous Audiometry

The otologist who is used to octave audiometry may claim that he is satisfied with the information he gets from this method. However, what he misses is only known to the otologist who uses continuous audiometry as a routine procedure. The latter

TABLE 1.—*Average Standard Deviation in Ten Normal Ears*

| Frequency (kilocycles per second) | 0.125 | 0.250 | 0.500 | 1 | 2 | 4 | 8 | Average |
|---|---|---|---|---|---|---|---|---|
| Continuous audiometry (db.) | 1.19 | 1.21 | 1.08 | 0.55 | 0.38 | 1.48 | 2.27 | 1.17 |
| Octave audiometry (db.) | 3.31 | 2.04 | 2.02 | 1.66 | 1.73 | 2.16 | 3.18 | 2.43 |
| Mean of 16 frequencies in continous audiometry | | | | | | | | 1.41 |

TABLE 2.—*Average Standard Deviation in Ten Impaired Ears*

| Frequency (kilocycles per second) | 0.125 | 0.250 | 0.500 | 1 | 2 | 4 | 8 | Average |
|---|---|---|---|---|---|---|---|---|
| Continuous audiometry (db.) | 2.77 | 2.83 | 2.87 | 1.55 | 1.64 | 2.10 | 3.38 | 2.41 |
| Octave audiometry (db.) | 3.29 | 3.60 | 4.12 | 3.47 | 2.50 | 3.97 | 4.53 | 3.68 |
| Mean of 16 frequencies in continous audiometry | | | | | | | | 2.72 |

TABLE 3.—*Average Standard Deviation at Sixteen Different Frequencies in Continuous Audiometry of Eight Ears Presenting Conductive Deafness and Nine Ears Presenting Perceptive Deafness*

| Frequency (kilocycles per second) | 0.125 | 0.250 | 0.500 | 1 | 1.5 | 2 | 2.5 | 3 | 3.5 |
|---|---|---|---|---|---|---|---|---|---|
| Conductive deafness (db.) | 3.48 | 3.55 | 2.08 | 2.17 | 1.97 | 2.02 | 3.39 | 3.07 | 3.51 |
| Perceptive deafness (db.) | 2.46 | 2.37 | 4.18 | 1.64 | 1.86 | 2.85 | 1.53 | 2.07 | 3.51 |

| Frequency | 4 | 5 | 6 | 7 | 8 | 9 | 10 | Average |
|---|---|---|---|---|---|---|---|---|
| Conductive deafness | 3.65 | 3.52 | 3.26 | 2.89 | 3.34 | 3.05 | 1.93 | 2.93 |
| Perceptive deafness | 1.85 | 4.61 | 4.35 | 3.67 | 2.69 | 3.08 | 2.89 | 2.89 |

Conclusion: In these two groups of hearing impairment a significant difference in standard deviation does not exist.

will be fascinated by the wealth of details (Fig. 3), the pronounced character of typical audiograms (Fig. 4), and the very high fidelity in repeated determinations.

## Continuous Audiometry in Children

A child can much more easily make a determination about hearing or not hearing a sweep tone with its changing pitch and loudness than about hearing or not hearing a tone of fixed frequency which changes in loudness only. Bunch (1943) compared a sweep tone to the visual impression roused by a hare running through a landscape.

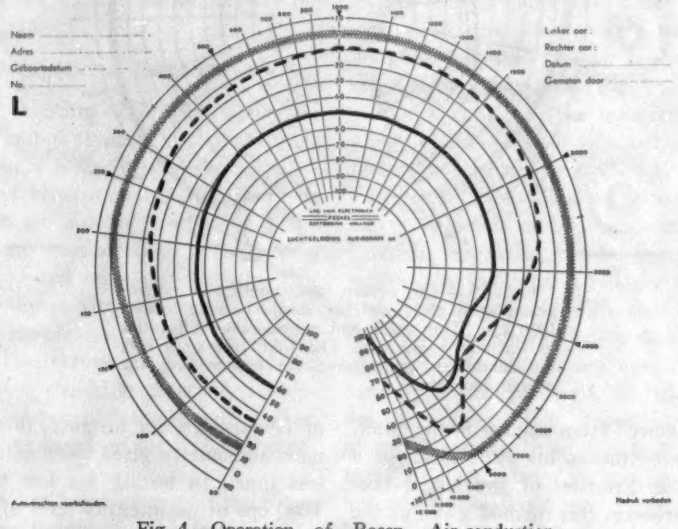


Fig. 4.—Operation of Rosen. Air-conduction audiogram. The solid line represents hearing before the operation; the broken line, hearing after the operation.

How much more easily can we detect such a moving hare than a stationary one! Moreover, the child is much more interested in a tone of changing pitch, which may remind him of a bird or music, than in a meaningless fixed pure tone.

The technique in children is somewhat different from that in adults. We move the frequency dial back and forth over small areas, first on high intensity levels, later, possibly in the next session, on lower ones. We record the results as little lines on the audiocard. If necessary, play audiometry is used. In this way the child can be tested at an earlier age than with other techniques.

In children receiving hearing tests on both ears the "switch-over method" proved to be of great help. The child is very much impressed by the sudden jumping-over of irregularly and without the child noticing that we are doing anything.

In this way it is possible to obtain good information on the hearing pattern of the child. We are able to determine at which frequency hearing diminishes critically and what is the slope of the hearing loss. With this information we can better understand why one child has less difficulty in hearing and pronouncing the consonants than another child, though their octave audiograms may be identical (Figs. 5A and 5B).

**Screening Tests in Children and Adults**

The detection of hearing defects in great numbers of school children, soldiers, and laborers requires a simple, quick, and accurate technique. In octave audiometry this is usually achieved by testing on an intensity above the threshold and on a limited number

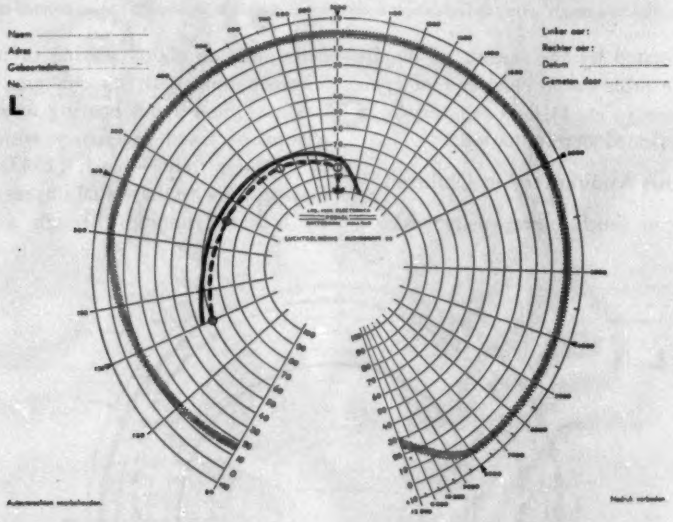

Fig. 5A.—The octave and continuous audiograms of a child with an abrupt hearing loss at 1000 cps. This child had considerable difficulties in learning consonants. The solid line represents the continuous audiogram; the broken line, the octave audiogram.

the "bird's voice" from one ear to the other, and usually he moves his eyes, head, or a finger in the direction of the sound perceived. Moreover, this method gives us the certainty that a tone is heard. Of course, the switch-over from the tone must be done of frequencies, for instance, three. Continuous audiometry gives more information in less time. In testing for low tones under 1000 cps of an intensity level of 20 db. and for high tones on a level of 15 db., hearing can be determined from 200-8000 cps in

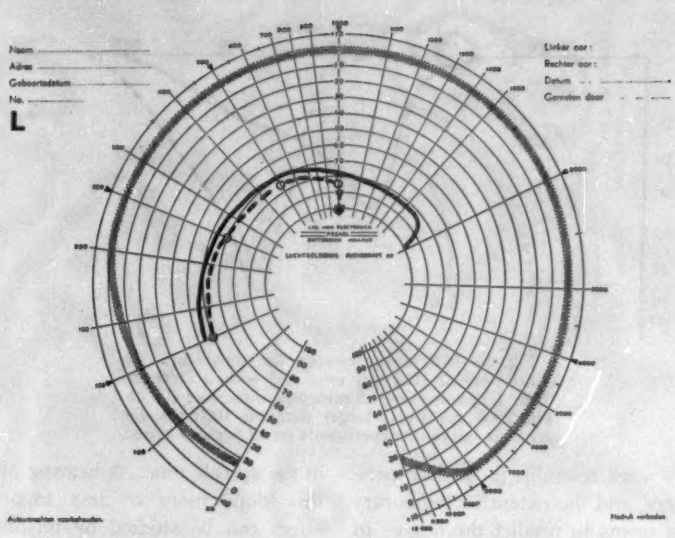

Fig. 5B.—The octave and continuous audiograms of another child. The octave audiogram shows an abrupt hearing loss at 1000 cps. This child had less difficulty in learning the consonants. The solid line represents the continuous audiogram; the broken line, the octave audiogram.

less than one minute. When earphones with rubber cuffs are used, normal room noise not surpassing 40 db. will not be disturbing (Spoor, 1954). This is of great importance, because a sound-treated room is seldom available. Thus, continuous audiometry has many advantages.

### Occupational Deafness

Repeated checking of hearing acuity for high tones and thus early detection of an acoustic trauma is the best method for the prevention of occupational deafness. In its initial stages the acoustic trauma is a dip. These dips are nominated by their deepest point. It appeared from our statistics that such a dip may be situated at each part of the basilar membrane between 2000 and 7000 cps. The erroneous notion that the 4000 cps region should be especially susceptible is an understandable misinterpretation of observations based on the incomplete octave audiogram.

With the sweep-frequency technique every acoustic trauma will be detected and its exact pattern registered in a very short time. This holds true especially for the experimental acoustic trauma with pure tones, which is the basis of our knowledge of the damaging effect of noise on man (van Gool, 1952). The factors intensity, duration, and frequency could be studied separately, and the recovery of a trauma could be followed, even in as short steps as one-fourth of a minute (Fig. 6).

In the same way the traumatic effect of a high or low pitched, an equal or a pulsing factory noise could be studied in the laboratory (Rol, 1956). This was done by imitating these noises or by registering them on tape. In comparing the fatiguing effect of octave bands, filtered from the total noise, with pure tones, no difference as to the effect of pitch and intensity was found. A study of occupational deafness in the factories and dockyards of the isle of Dordrecht suggests that acoustic trauma is acquired essentially in the first month of exposure to noise, the increasing of a hearing loss during continued exposition being largely due to presbyacusis (van Leeuwen, 1955). Audiograms of newcomers taken

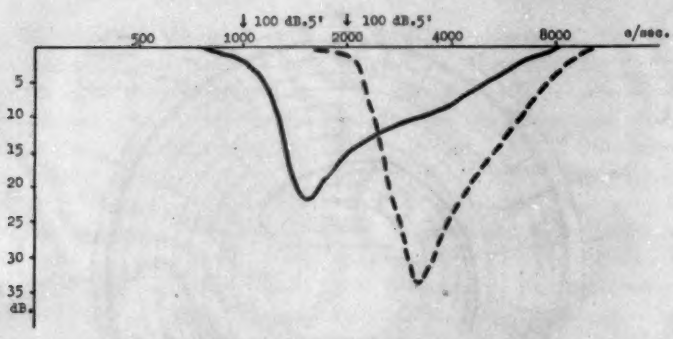


Fig. 6.—Difference between the tonal dips obtained with a 1000 cps tone and with a 2000 cps tone. Exposure time: 5 minutes. Intensity: 100 db. The 2000 cps dip is larger than the 1000 cps dip. Average curve of experiments on 12 persons tested.

after a day's work resemble those of experienced workers, and the extent of temporary hearing loss seems to predict the degree of permanent loss to be expected from continued exposure.

### Speech Validity Percentages

Where the hearing capacity for speech is concerned, the highest accuracy is necessary. The evaluation of hearing losses, according to the tables of Fowler, is based on four values, namely, 500, 1000, 2000, and 4000 cps, obtained by octave audiometry. The main objections to this procedure are that the hearing capacity for the intermediate frequencies is neglected and that the probable error of octave audiometry is higher than that of continuous audiometry (Tables 1, 2, and 3). Scientifically more justified validity percentages can be obtained from different calculations based on the mean values of three continuous audiograms. The calculations should not only take into account the four values mentioned, but corrections based on the slope of the curve should be made. Thus the results may differ considerably. A calculation of the probable error should be added.

### Continuous Audiometry and the Choice of a Hearing Aid

A continuous audiogram will inform us in detail about the slope of the hearing curve in the speech zone. A hearing aid may alter this slope more or less favorably. This effect can be studied by making a second audiogram, in which the audiometer tones reach the patient through his hearing aid (Fig. 7). This is realized by placing the headphone of the audiometer on the microphone of the hearing aid.

### Comment

The von Békésy audiometer (1947) realizes the principle of continuous audiometry in another way. The tonal scale is presented mechanically at a certain speed to the patient, and he is required to maintain the intensity at the threshold. A disadvantage of this method is the long time needed for complete bone- and air-conduction audiograms for both ears. A second one is the incapacity of patients to follow abrupt hearing losses. Thus the contour of a dip or abrupt hearing loss will be considerably altered, and often quite erroneous curves are obtained because the patient is absolutely at a loss about his threshold. The third disadvantage is the high cost of the apparatus, making it obtainable only by large clinics. As an advantage, the greater objectivity realized by the equal speed in which the tonal scale is transgressed is mentioned. In our opinion, this is a disadvantage, as too much time is lost on unimportant parts of the audiogram, whereas difficult parts are passed by relatively too fast.

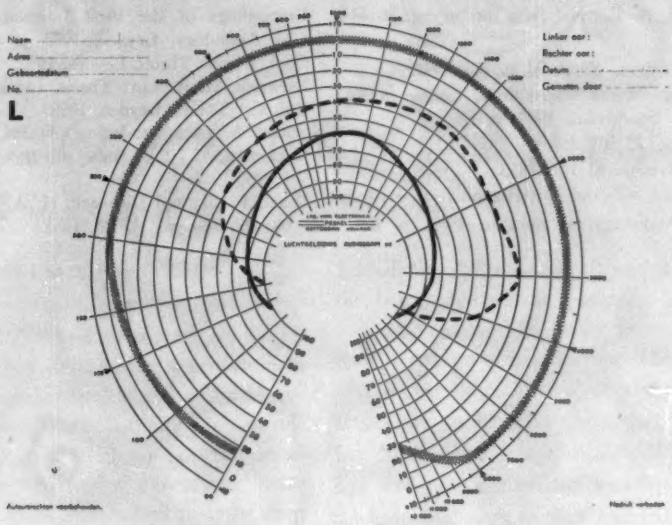

Fig. 7.—Checking the effect of a hearing aid on the threshold: The optimum result is obtained when the hearing-aid audiogram is straight in the speech area, as it appears in this patient. The solid line indicates the audiogram without the hearing aid; the broken line, the audiogram with the hearing aid.

On the contrary, in our technique the speed of passing the tonal scale is adapted to the slope of the audiogram. As shown in Figure 2, each following procedure is a consequence of the foregoing one. Thus much time is saved, and the attention of the operator is automatically focused on the more important parts. He can check his findings very easily and thus increase the exactness of his measurements. Compared with the octave audiometry technique, this procedure is more agreeable for the operator, as his active cooperation is more intense and more useful.

## Summary

In continuous, or sweep-frequency, audiometry, the tonal scale is presented to the patient on a fixed intensity level. Thus horizontal sections are obtained through the audiogram, revealing the exact contour of all hearing losses. The decisions of the patient about hearing or not hearing the sweep tone are directly noted on an audiocard fixed to the audiometer. Thus much time is saved. Masking in continuous audiometry is more accurate, as the intensity of the masking noise can be easily adjusted to the level of the test tone.

As the output of the audiometer is adapted from 200-8000 cps to the normal zero isophone, this apparatus is very suitable for screening tests in school children, in factory workers, and in soldiers. In fact, the early detection and exact registration of an acoustic trauma is only possible in this way.

In comparison with other techniques, continuous audiometry gives more and exacter information in less time. It should be called "high-fidelity" audiometry.

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# Fenestra Ovalis for Otosclerotic Deafness

## An Adjunct to Stapes Mobilization

SAMUEL ROSEN, M.D., New York

In April, 1952, and in several communications since,* I described a new technique for stapes mobilization which restored the hearing in otosclerotic patients from levels of 50-60 db. loss to 30 db. and up to normal hearing levels. With few exceptions, these patients have since maintained their hearing improvements. The first patient, whose rigid stapes was mobilized April 3, 1952, attained a hearing level of 0-5 db., which has been maintained to date.

A comprehensive report was made in 1955 [8] on this work, with an analysis of results, indications, audiological findings, functional goals, etc. Since 1952, many distinguished otologists here and abroad, using the Rosen technique in about 8000 cases,† have corroborated these findings.

In the Rosen technique of stapes mobilization, the lower half of the drum (and adjoining skin) is lifted out of its sulcus, folded upward upon itself, and 2-3 mm. of the bony edge of the posterior bony canal wall are removed in order to uncover and widely expose the hidden ossicles (particularly the long process of the incus, the stapes, its tendon, the crura, and footplate) for direct visual manipulation (Figs. 1 and 2). Pressure against the anterior aspect of the stapedial neck is then exerted in a posterior direction with the mobilizer until the fixed footplate is rendered freely mobile (Fig. 3C). Stapes mobilization preserves nature's own pathway of sound transmission via the intact ossicular chain, through the oval window to the cochlea.

This original technique of stapes mobilization may be referred to as the "indirect" method, since the force which is applied to the neck of the stapes is transmitted to and moves the footplate. But when the bony otosclerotic mass causes extreme fixation of the footplate, continued pressure against the stapedial neck may not mobilize the

Accepted for publication June 12, 1956.

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† References 9-11. Werth, R.: A New Operation for Otosclerosis: Rosen Mobilization of Stapes, read before the Third Turkish Otorhinolaryngology Congress, Istanbul, Sept., 1955. Clerc, P.: Mobilisation de l'étrier— Operation de Rosen: Commentaires sur 52 cas, read before the 53rd French Congress of Otorhinolaryngology, Paris, Oct., 1955. Lindsay, J. R.; Goodhill, V.; Kos, C.; Shambaugh, G.; House, H.; Meltzer, P., and Fowler, E. P., Jr.: Symposium on the Operation for the Mobilization of the Stapes in Otosclerotic Deafness, read before the American Laryngological, Rhinological and Otological Society, Montreal, Canada, May 17, 1956. Personal communications to the author: (America) F. Altman, R. Bellucci, G. Boyd, S. Baron, L. Coleman, E. Fowler, H. House, C. Kos, D. Myers, B. Ronis, J. Sataloff, G. Shambaugh, J. Shea Jr., Silcox S. Sander; (Germany) H. Wullstein, F. Zollner; (England) T. Cawthorne, F. C. Ormerod, Mr. Ventner; (Scotland) I. Simson Hall; (France) H. Guillon, R. Maspetiol, M. Pruvot, G. Portman; (Italy) M. Arslan, L. Pietrantoni, L. Bocca; (Israel) R. Werth, Dr. Rubenstein; (Greece) T. Demetriades, R. Yannoulis; (Turkey) S. Karatay; (Lebanon) J. Marini; (Spain) Antoli-Candela, Scola; (Switzerland) L. Reudi; (Sweden) T. Skoog, P. Frenckner, L. Holmgren, M. Diamant; (Argentina) J. Tato; (Holland) H. V. Dishoek.

Fig. 1.—When drum and skin are reflected upward, too little of the stapes can be seen and mobilization cannot be performed.

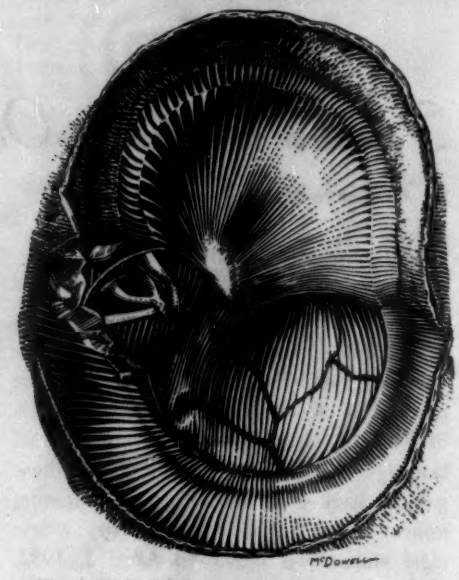

Fig. 2.—After 2-3 mm. of the very edge of the posterior bony canal wall just external to the incus and stapes is removed, a full view of the long process of the incus, the incudostapedial junction, the head, neck of the stapes, and the entire length of the stapedial tendon is obtained. This exposure is absolutely necessary for stapes mobilization.

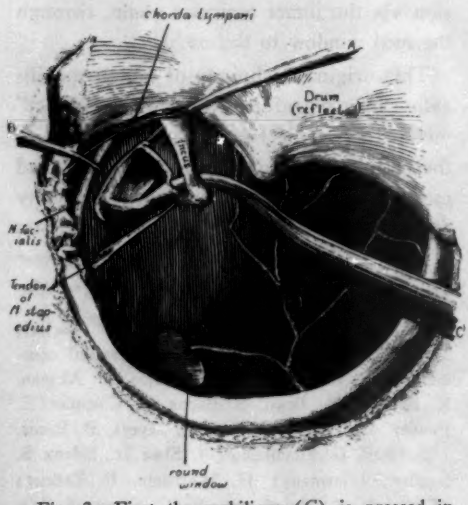


Fig. 3.—First the mobilizer (*C*) is pressed in a posterior direction against the neck of the stapes (indirect method) in the line of the crura or in the line of the stapedial tendon when the crura are not distinctly seen. When this maneuver fails to mobilize the footplate the explorer is used at *A* and *B* in the positions indicated to pry loose the rigid footplate.

footplate but may instead fracture the crura, leaving behind the intact rigid footplate. For such cases, a further development of the stapes mobilization technique was devised,‡ by applying the mobilizing force directly to the footplate itself, the site of the pathological fixation. This is called the "direct" method of stapes mobilization. In the "direct" method, the sharp-pointed explorer (Fig. 4) is used to exert the necessary pressure against the peripheral margin of the fixed footplate to pry it loose and render it mobile. At various sites, the point of the explorer is gently wedged in between the bony margin of the oval window and the peripheral margin of the stapedial footplate for a distance no greater than the thickness of the footplate, so as not to enter the vestibule (Fig. 3*A* and *B* and Fig. 5). By mobilizing the footplate itself, the hearing has been restored to the same high levels as by the indirect method.[12]

It has been established that all patients suitable for fenestration are equally suitable

‡ References 12 and 13.

Fig. 4.—The mobilizer, left, used against the neck of the stapes, and the explorer, right, the point of which is used to pry the footplate loose in the direct footplate mobilization.

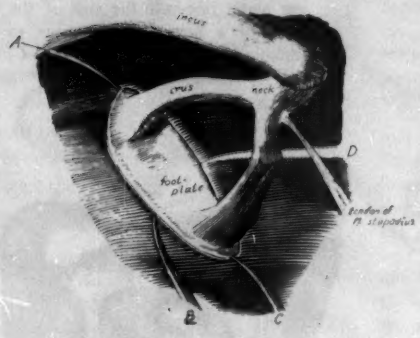


Fig. 5.—*A* and *B* show the pointed explorer used anteriorly and inferiorly to mobilize the footplate. Occasionally the explorer is used at the posterior edge of the footplate (*C*) and rarely at the anterior edge (*D*).

for mobilization. In addition, those patients with a hearing loss of about 30 db., who are not yet suitable for fenestration or a hearing aid, are suitable for mobilization, and, finally, those with profound deafness can be improved sufficiently by mobilization to allow for successful use of a hearing aid. The spectrum of handicapped patients who can be rehabilitated for aided or unaided hearing by stapes mobilization is much wider than hitherto possible with any type of surgery.

When stapes mobilization and stapedectomy, which were widely employed from 1876 to 1900 by Kessel,[14] Miot,[15] Blake,§ Jack,‖ and others,¶ were abandoned over 50 years ago as worthless and dangerous,# necessity then became the mother of invention. Attention was next focused on making a labyrinthine fistula in the external semicircular canal which provided an altogether different and less direct route for the passage of sound to the cochlea than that provided by nature through the oval window. Passow,[29] Jenkins,[30] Bárány,* Holmgren,[31] Sourdille,[32] and Lempert[33] were the pioneers in the development of fistulization, or fenestration, of the labyrinth for otosclerotic deafness. Sourdille in 1937 described his multistage fenestration technique at the New York Academy of Medicine. In 1938, Lempert described his one-stage fenestration, which proved to be a practical and noteworthy advance in the surgery of otosclerotic deafness.

Nothing was heard of stapes mobilization from 1900 until 1952, when I described my technique. In all likelihood, the old technique of stapes mobilization (1876-1900), which was performed through a myringotomy, had to be abandoned mainly because with their approach they could see little or none of the stapes in over 85% of cases. It is interesting to speculate on how the surgery of otosclerosis might have developed had the present technique of stapes mobilization been employed 50 years ago.

The purpose of this communication is to present still another technique for the relief of otosclerotic deafness—the creation of a fenestra in the oval window, which allows the passage of sound directly through the oval window to the cochlea.

This fenestra ovalis is made through the inferior peripheral margin of the footplate

§ References 16-18.

‖ References 19-22.

¶ References 15 and 24-26. Michel, U., cited by Miot.[15]

# References 27 and 28.

* Bárány, R., in discussion on Jenkins,[30] p. 617.

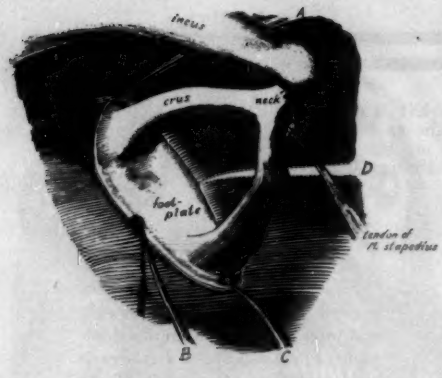


Fig. 6.—Fenestra ovalis at the inferior margin of the footplate created by penetrating the explorer (*B*) for a half-millimeter into the vestibule, leaving a circular opening about one-half the size of the head of a pin, after attempts to mobilize the footplate at *A*, *C*, and *D* failed.

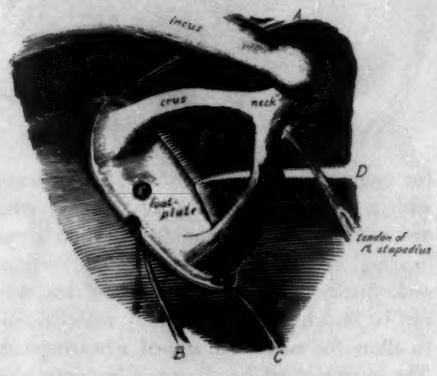


Fig. 7.—*E* indicates the site of the fenestra ovalis through the body of the footplate.

at the junction with the bony margin of the oval window, or through the center of the footplate itself (Figs. 6 and 7). The fenestra of the oval window is usually made by penetrating the explorer into the vestibule for a half-millimeter. The explorer is held between the index finger and thumb and is turned clockwise and counterclockwise several times in a circular fashion as it penetrates gently, like boring an opening into the vestibule (vestibulotomy). When the explorer is removed, the circular opening made by the explorer can be seen leading into a dark-looking cavity, i.e., the vestibule. In many instances perilymphatic fluid can be seen to escape. Vertigo may be absent altogether or may occur for a few hours or occasionally for a few days. Almost all the patients leave the hospital within 24 hours after operation. Rarely does the vertigo hospitalize the patient more than one additional day. In many cases following fenestration ovalis, the hearing has been restored to the same high levels as those achieved by the indirect or direct mobilization techniques.

Fenestration of the oval window was first done on the side of an unsuccessful fenestration nov-ovalis performed by another surgeon in 1950 and revised by him in 1951. In August, 1954, in an attempt to improve the hearing in this patient, a small segment of the inferior edge of the footplate was fractured away, leaving a good-sized opening of the oval window, through which one could see into the vestibule. Perilymph escaped. The hearing in this case improved 15 db.

Subsequent attempts to create such a fenestra in the oval window were made in virgin ears after attempts at mobilization at the neck or at the footplate itself failed to improve the hearing. Following fenestration of the oval window the hearing improved from preoperative levels of 60 db.

to as high as 15 db. or better by pure-tone and speech audiometry.

The following successful cases are being reported at this time to demonstrate the feasibility of this technique. In some of the illustrative cases, several months have elapsed from the time of operation to the last audiological tests. In many other cases there has been marked hearing improvement, but since the last audiological tests followed too shortly after operation and complete audiological data are not yet available, these are not being reported here. In other cases, the hearing did not improve. An analysis of all the cases will be made later.

In the first series of cases reported below, the fenestra ovalis was performed after the crura were fractured during mobilization. In the second series of five cases, although mobilization failed, the crura remained intact.

CASE 1.—A 51-year-old man who had bilateral progressive deafness for over 15 years. There was a constant ringing tinnitus present in both ears. The preoperative hearing loss by pure tone and speech was 45 db. in the right ear and 40 db. in the left. On Dec. 1, 1955, the patient had a mobilization operation. The left stapes was rigid and the crura exceedingly thin. Attempted mobilization at the stapedial neck caused the crura to fracture, and the hearing did not improve. The point of the explorer was then inserted between the bony ridge of the oval window and the inferior edge of the footplate (Fig. 6). The explorer penetrated the area for about 0.5 mm. with a clockwise and counterclockwise circular motion during penetration. When the explorer was removed, the circular opening could be seen in the penetrated area, half the size of the head of a pin, through which one could see into the vestibule. Perilymph was seen to escape from the circular fenestra of the oval window. Three months after the operation, his hearing reached the 15 db. level for pure tone and speech. There was no postoperative vertigo (Fig. 8).

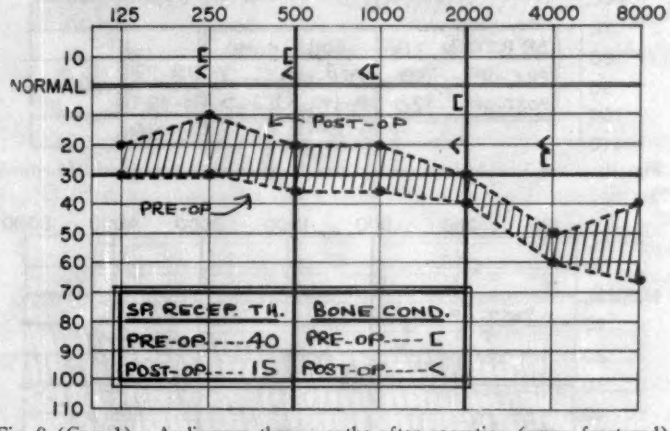


Fig. 8 (Case 1).—Audiogram three months after operation (crura fractured).

CASE 2.—A 43-year-old woman who had a history of bilateral progressive deafness for 18 years. There was a history of family deafness, and she heard better in the subway. She had constant ringing tinnitus in both ears and wore a hearing aid on the right side. Her preoperative hearing loss by pure-tone and speech audiometry was 65 db. On Aug. 8, 1955, her right stapes was found to be very rigid and continued pressure against the anterior aspect of the neck finally caused both crura to fracture, leaving the rigid footplate. The explorer was then placed between the bony ridge of the oval window and the inferior edge of the footplate (Fig. 6). The same procedure for creating the fenestra ovalis was followed. When the explorer was removed, the circular opening was seen in the penetrated area, through which one could see into the dark vestibule. Perilymph was seen to escape from the circular fenestra of the oval window. Five months after the operation, the hearing reached the 20 db. level by pure-tone and speech audiometry (Fig. 9).

CASE 3.—A woman of 39 with a history of bilateral progressive deafness for over 10 years. Her father had been deaf for many years. She

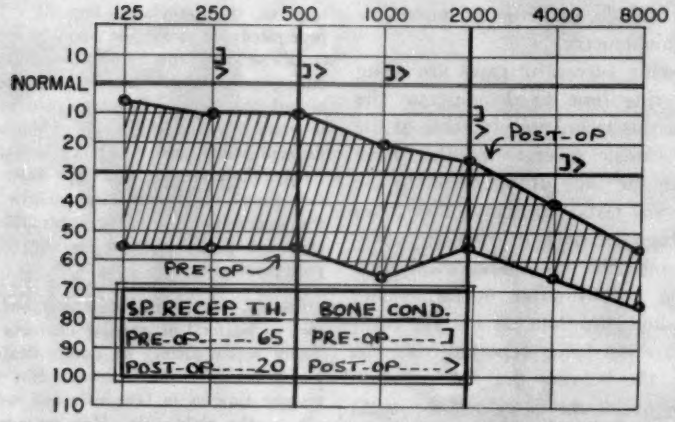


Fig. 9 (Case 2).—Audiogram five months after operation (crura fractured).

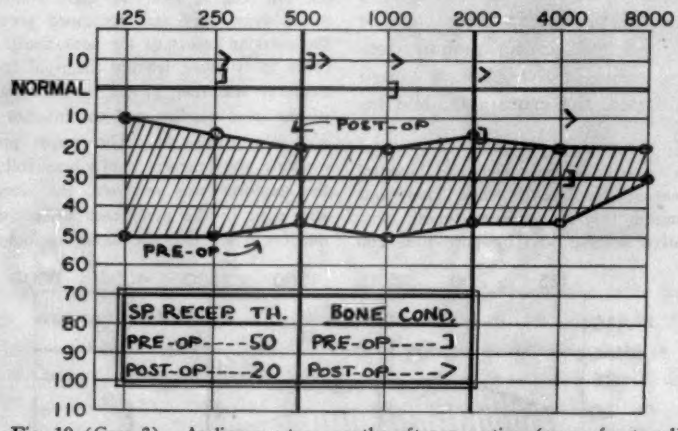


Fig. 10 (Case 3).—Audiogram two months after operation (crura fractured).

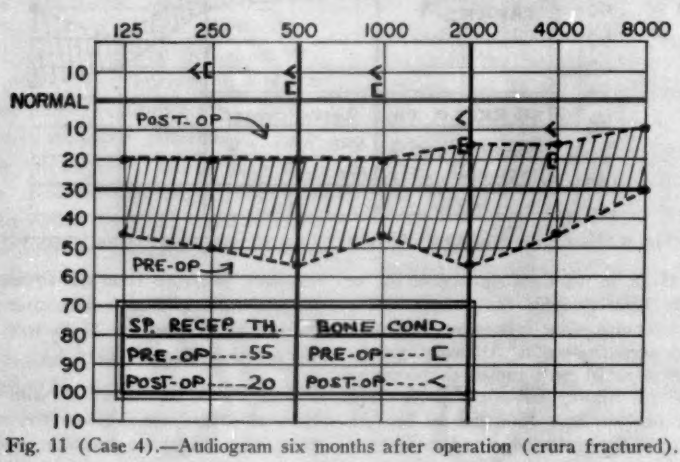


Fig. 11 (Case 4).—Audiogram six months after operation (crura fractured).

heard better in the subway and had intermittent tinnitus in both ears. She wore a hearing aid in the right ear for six years. The preoperative hearing loss for pure tone and speech was 50 db. On March 2, 1956, her right stapes was found very rigid and continued pressure against the anterior aspect of the neck finally caused both crura to fracture, leaving the rigid footplate. The point of the explorer was then inserted between the bony ridge of the oval window and the inferior edge of the footplate (Fig. 6). The same procedure for creating the fenestra ovalis was followed as in the previous cases. When the explorer was removed, the circular opening could be seen in the penetrated area, through which one could see into the dark vestibule. Perilymph was seen to escape from the circular fenestra of the oval window. Two and a half months after the operation, her hearing reached the 20 db. level for pure tone and speech (Fig. 10).

CASE 4.—A 43-year-old woman with a history of bilateral progressive deafness of five years' duration. There was constant clicking tinnitus in her left ear. The preoperative hearing loss by pure-tone and speech audiometry was 55 db. On Dec. 1, 1955, the patient had a mobilization operation. The left stapes was rigid, and attempted mobilization at the neck caused the crura to fracture without improving the hearing. The point of the explorer was then inserted between the bony ridge of the oval window and the inferior edge of the footplate (Fig. 6). The same procedure for creating the fenestra ovalis was followed. When the explorer was removed, the circular opening could be seen in the penetrated area, through which one could see into the dark vestibule. Perilymph was seen to escape from the circular fenestra of the oval window. Six months later her hearing was at the 20 db. level for pure tone and speech (Fig. 11).

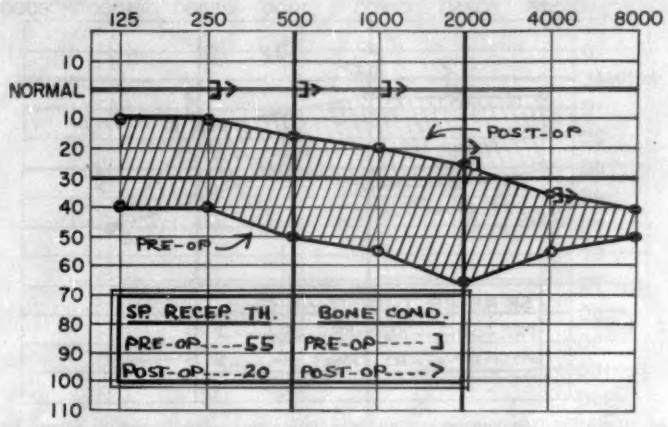


Fig. 12 (Case 5).—Audiogram nine months after operation (crura fractured).

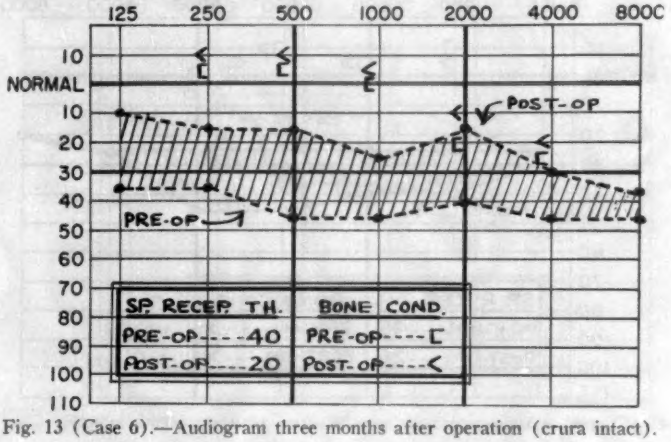


Fig. 13 (Case 6).—Audiogram three months after operation (crura intact).

CASE 5.—A woman of 34 with a history of bilateral progressive deafness for 12 years. There was intermittent tinnitus in both ears. The preoperative hearing loss for pure tone and speech was 55 db. On July 19, 1955, the right stapes was found to be very rigid and the crura fractured when pressure was continued against the anterior aspect of the neck of the stapes. The explorer was then applied to about the center of the body of the footplate and penetrated the area for about a half-millimeter with a clockwise and counterclockwise circular motion (Fig. 7). When the explorer was removed, the circular opening could be seen in the penetrated area, through which one could see into the dark vestibule. Perilymph was seen to escape from the circular fenestra of the oval window. Nine months after the operation her hearing was at the 20 db. level by pure-tone and speech audiometry (Fig. 12).

CASE 6.—A 54-year-old mother of two children with a history of progressive bilateral deafness for five years. There was constant tinnitus in the left ear. Her preoperative hearing loss by pure-tone and speech audiometry was 40 db. On Dec. 13, 1955, the left stapes was found to be extremely rigid. Great pressure on the stapedial neck resulted in no movement at all of the stapes or stapedial tendon. The crura did not fracture. The explorer was applied at about the center of the body of the footplate and penetrated the area for a half-millimeter with a circular clockwise and counterclockwise motion (Fig. 7). When the explorer was removed, the circular opening could be seen in the penetrated area, through which one could see into the dark vestibule. Perilymph was seen to escape from the circular fenestra of the oval window. Three months after the operation, the hearing was at the 20 db. level for pure tone and speech (Fig. 13).

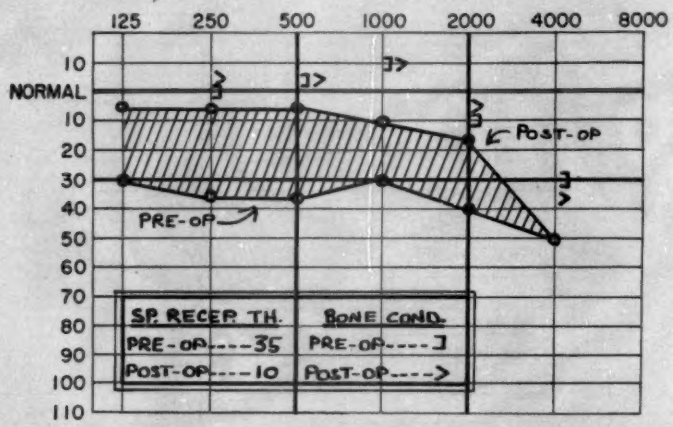


Fig. 14 (Case 7).—Audiogram one and one-half month after operation (crura intact).

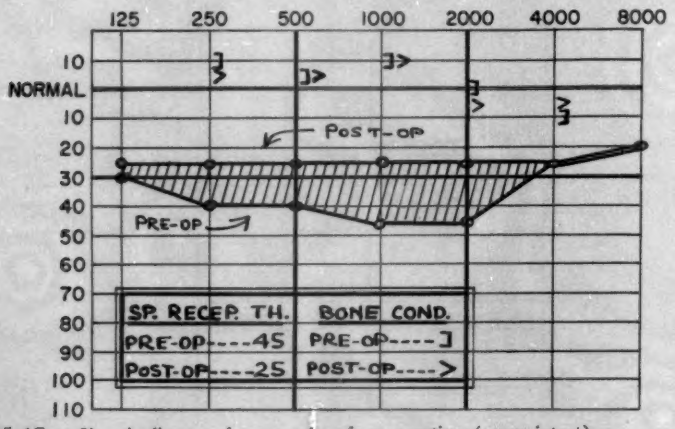


Fig. 15 (Case 8).—Audiogram four months after operation (crura intact).

CASE 7.—A 37-year-old man with a history of bilateral progressive deafness for 18 years. He had intermittent tinnitus in the left ear and heard better in the subway. He had been wearing a hearing aid for one year. His preoperative hearing loss by pure-tone and speech audiometry was 35 db. On Aug. 8, 1955, the right stapes was found to be extremely rigid and repeated pressure on the neck of the stapes failed to mobilize the footplate. The crura remained intact. The point of the explorer was then inserted between the bony ridge of the oval window and the inferior margin of the footplate (Fig. 6). The explorer penetrated the area for about a half-millimeter with a clockwise and counterclockwise circular motion. When the explorer was removed, the circular opening could be seen in the penetrated area, through which one could see into the dark vestibule. Perilymph was seen to escape from the circular fenestra of the oval window. Six weeks after operation, the hearing was at the 10 db. level for pure tone and speech (Fig. 14).

CASE 8.—A 36-year-old mother of two children had bilateral progressive deafness for 12 years. Her mother had been deaf for a number of years. The patient had intermittent buzzing tinnitus. Her preoperative hearing loss by pure-tone and speech audiometry was 45 db. On July 6, 1955, the right stapes was found to be very rigid and extreme pressure on the neck of the stapes failed to mobilize it. The crura remained intact. The explorer was then placed at about the center of the body of the footplate and penetrated the area for a half-millimeter with a clockwise and counterclockwise circular motion (Fig. 7). When the explorer was removed, the circular opening could be seen in the penetrated area, through which one could see into the dark vestibule. Perilymph was seen to escape from the circular fenestra of

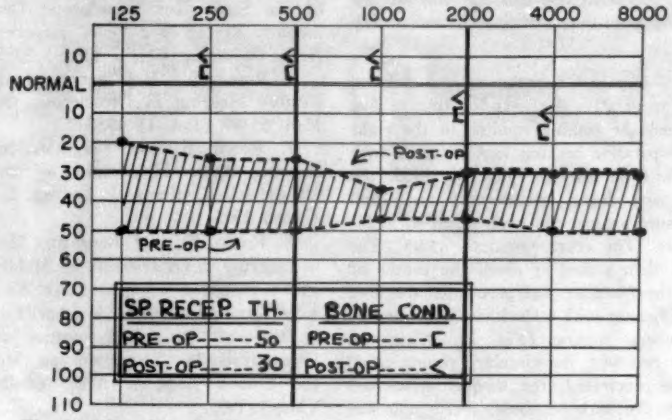


Fig. 16 (Case 9).—Audiogram five months after operation (crura intact).

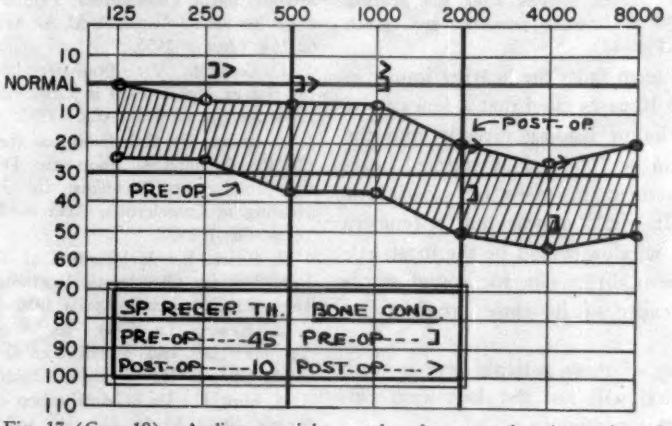


Fig. 17 (Case 10).—Audiogram eight months after operation (crura intact).

the oval window. Five months later the hearing was at the 25 db. level by pure-tone and speech audiometry (Fig. 15).

CASE 9.—A 35-year-old mother of two children complained of bilateral progressive deafness for 15 years with intermittent tinnitus. She wore a hearing aid. The preoperative hearing loss for pure tone and speech was 50 db. On Dec. 15, 1955, the left stapes was found to be very rigid and extreme pressure on the stapedial neck failed to mobilize the stapes although the crura remained intact. The explorer was then placed at about the center of the body of the footplate and penetrated for a half-millimeter with a clockwise and counterclockwise circular motion into the vestibule (Fig. 7). When the explorer was removed, the circular opening could be seen in the penetrated area, through which one could see into the dark vestibule. Perilymph was seen to escape from the circular fenestra of the oval window. Five months after operation, the hearing was at the 30 db. level by pure-tone and speech audiometry (Fig. 16).

CASE 10.—A 47-year-old mother of two children had bilateral progressive deafness for four to five years with constant ringing tinnitus in the right ear. Her preoperative hearing loss for pure tone and speech was 45 db. On July 26, 1955, the right stapes was found to be very rigid and extreme pressure against the neck failed to mobilize the stapes. The crura remained intact. The explorer was then placed at about the center of the body of the footplate and penetrated the area for a half-millimeter with a clockwise and counterclockwise circular motion (Fig. 7). When the explorer was removed, the circular opening could be seen in the penetrated area, through which one could see into the dark vestibule. Perilymph was seen to escape from the circular fenestra of the oval window. Eight months later the hearing was at the 10 db. level by pure-tone and speech audiometry (Fig. 17).

It would seem from the hearing improvement in the 10 cases cited that a fenestra of the oval window leading directly into the vestibule can be followed by marked hearing improvement in cases of otosclerotic deafness. It would appear that a fenestra in the oval window would be the most natural and most direct site for sound transmission because of its close proximity to the cochlea.

Follow-up of these patients over a much longer period will test the long-term efficiency of this procedure.


## Summary

A fenestration of the oval window (fenestra ovalis) for otosclerosis is described with 10 illustrative cases.


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# Case Reports

## RHABDOMYOSARCOMA OF HYPOPHARYNX

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E. B. GAMMELL, M.D.
and
WALTER THAYER, M.D., Providence, R. I.

THE PRESENCE of tumors of mesodermal origin in the head and neck is not unusual. However, a rhabdomyosarcoma is unusual if one is to search the literature for case reports. These tumors are few in number and have been reported primarily in the tongue, cheek, palate, tonsil, nose, and nasal septum. The presence of a rhabdomyosarcoma in the hypopharynx seems unusual enough to warrant its addition to the literature.

The rhabdomyosarcoma is a highly malignant tumor which spreads by local infiltration and distant metastases but rarely involves any of the regional lymph nodes, although one case reported by Davis and Hare had regional lymph-node involvement.[5] It is a malignant tumor which is composed in large part of striated muscle, usually of embryonal type, and sometimes contains immature muscle cells (myoblasts). There is a tendency to recurrence which is greater than average. The tumor is quite rare and usually affects the voluntary muscle but can also occur in the involuntary muscle of the stomach and urinary tract. The disease is usually fatal, recurring after local excision with metastases to the lungs. Histologically there is proliferation of broad ribbon-like cellular processes with beading or cross striation and bizarre binucleated forms usually embedded in a pale myxomatous matrix. Pastore, Sahyoun, and Mandeville report a rhabdomyosarcoma of the maxillary antrum.[1] Thorek and Neiman report a rhabdomyosarcoma of the esophagus and review the world literature. They found 58 sarcomas of the esophagus, of which only 3 were rhabdomyosarcomas.[2] This particular tumor is commoner in the heart, the vagina, the uterus, the bladder, the testes, and the kidney. The tumor in their own case, which was successfully removed, was found in the region of smooth musculature in the thoracic esophagus. Dargeon and Stobbe report on embryonal rhabdomyosarcoma of the head and neck in children and adolescents.[3] Fifteen cases were reported from the Memorial Hospital in New York by them, and none were found to involve the pharynx. Schall and Cordray report unusual tumors of the upper respiratory tract and, in particular, a rhabdomyosarcoma in a 6-year-old boy on the posterior border of the septum.[4] Davis and Hare report in a child a case of an embryonal rhabdomyosarcoma in the nasopharynx.[5] This had been first mistaken for a juvenile fibroma of the nasopharynx but later was correctly diagnosed as an embryonal rhabdomyosarcoma. Delcourt and Dustin, in Brussels, report a rhabdomyosarcoma in the nasopharynx of a child with cervical and pulmonary metastases.[6] They suggested intrabronchial dissemination as a cause of the pulmonary metastases. Recently, St. John and Zung-Pah Woo have reported a rhabdomyosarcoma in the nasopharynx.[7]

Accepted for publication April 16, 1956.

### Report of Case

A 54-year-old Negro man was transferred to the Rhode Island Hospital from the State Sanatorium at Wallum Lake, where he had been a patient in March, 1955.

*Past History.*—In 1949, he had had a tuberculous empyema and a positive sputum was also reported. A decortication of his right lung with a phrenic nerve crush was done in that year. In October, 1951, x-rays showed two cavities in his chest with fluid levels. Between 1952 and 1954, he was at Wallum Lake, being treated for advanced pulmonary tuberculosis. At this time, he had a resection of his right lower lobe and of a

sinus tract in the right posterior lateral chest wall.

Just prior to his admission to Wallum Lake, in March, 1955, he had been picked up by an ambulance from this hospital after collapsing on the street. He was treated for a lobar pneumonia and for acute alcoholism. He was transferred to the sanatorium because of past history of tuberculosis. At that time he complained of a sore throat, but his physical examination showed no appreciable etiology. This soreness became worse after his transfer, and he was examined by direct laryngoscopy, at which time he was found to have a huge, smooth, spherical swelling involving the right arytenoid, with fixation of the right vocal cord. This was considered an extrinsic laryngeal tumor. Biopsy specimens taken showed an undifferentiated malignancy. He was then transferred back to the Rhode Island Hospital through the Tumor Clinic, where he was first seen by our service. Admission was advised.

On admission he denied any history of hoarseness or dysphagia or weight loss. He had had some slight coughing and hemoptysis in the past, which was apparently related to his tuberculous process.

*Physical Examination.*—Patient was a thin, 54-year-old Negro man in no apparent distress.

Temperature 100 F, pulse rate 90, respiration rate 22, blood pressure 104/76.

Positive physical findings included a markedly swollen right arytenoid with a fixed right vocal cord on indirect laryngoscopy. Only a few small, soft, submandibular nodes were felt. There were two well-healed thoracotomy scars in the right posterior chest wall. Heart sounds were of fair quality but with a systolic Grade 1 murmur at the lower end of the sternum and a reduplication of his second apical sound. Liver was palpable two fingers below the costal margin.

*Laboratory Report.*—Urinalysis was normal. He had a fasting blood sugar of 85%, blood urea nitrogen of 7%, but with a prothrombin time of only 35% and a BSP of 11% retention in 45 minutes, probably due to cirrhosis of the liver. His white cell count was 8300, with 60% polymorphonuclears, 37% lymphocytes, and 3% monocytes. CSR was 38.

*X-Rays.*—Barium swallow revealed an irregular filling defect in the right side of the barium column in the hypopharynx. The findings were reported consistent with neoplastic invasion of the right side of the hypopharynx.

*Course.*—While in the hospital awaiting surgery, patient continued to run a spiking fever up to 101 and 102 F every afternoon. Blood cultures and febrile agglutinins were negative. Three gastric washings for acid-fast bacteria were negative. Medical consultation suggested that tuberculosis activity, cirrhosis, or malignancy could be the reason for his pyrexia. Chest x-ray showed an extensive right thoracoplasty with no active parenchymal disease. Metastatic series were negative.

It was felt that the tumor was progressing, and, in spite of his fever, laryngectomy was deemed advisable, with the purpose of removing the possible focus of his fever as well as removing the tumor.

A repeat biopsy was now reported by the Institute of Pathology in the following manner: Microscopic section showed four small pieces, partially covered with intact stratified squamous epithelium. The tumor consisted of masses of bizarre connected-tissue cells with marked pleomorphism of the nuclei. The cytoplasm was abundant with elongated processes. Many cells were spindle-shaped. The stroma was fibrous, and the tumor had been diffusely infiltrated with acute inflammatory cells. Mitoses were seen occasionally. There was a suggestion of cross striation of the cytoplasmic processes. The appearance of tumor was that of a sarcoma, probably derived from striated muscle.

*Operation.*—A horseshoe-shaped incision through the skin, subcutaneous tissues, and platysma was made, starting up the level of the hyoid on each side, extending down along the anterior borders of the sternomastoids, and crossing the midline at the level of the third tracheal ring. The flap was elevated and the strap muscles divided and separated from the larynx. Once the larynx was skeletonized, there appeared a very large tumor which was 5 to 6 cm. in diameter, attached to the prevertebral fascia and obviously as part of hypopharynx rather than larynx. However, the involvement of the larynx was due to its juxtaposition, and while the larynx showed no actual invasion, there was a distinct interruption of function by the mere presence of the tumor. It was easy to separate the tumor from the prevertebral fascia but not from the larynx. A margin of about 1 cm. was allowed around all portions of the free edge of the tumor in order to include all possible areas of malignancy. Otherwise, a routine laryngectomy was carried out en bloc with the tumor. No attempt was made to do a radical neck dissection at this time because the tumor rarely involves neighboring nodes. After removal of the tumor mass and the larynx, a snake-like segment of mucosa remained, which was not over an inch in width and which was used to form a new pharynx. This was done around a plastic tube leading into the esophagus and provided a satisfactory pathway for the ingestion of food.

Postoperatively, the patient made a good recovery from the point of view of surgery. He had no fistula develop, he had good healing of his wound, and his tracheotomy was very satisfactory.

However, about one week following operation, he began again to spike fevers ranging up to 105 F as a daily occurrence. No specific cause could be found for the fever. He was given antibiotics empirically, but without results.

Three weeks following his operation, he suddenly fell while getting up, became unconscious, started Cheyne-Stokes respiration, and died within a matter of hours.

*Autopsy.*—At autopsy the following cause of death was established: a left spontaneous pneumothorax. The left lung was collapsed, showing evidence of massive atelectasis of all lobes. There were numerous pleural adhesions from pericardial adhesions. There were several pulmonary artery thromboses and infarction of the surrounding lung. No evidence of metastases was found, and his brain was negative.

## Summary

A case is reported of a 54-year-old Negro man who had clinical and microscopic evidence of a tumor consistent with rhabdomyosarcoma of the hypopharynx. The tumor was successfully removed, only to have the patient succumb to a pulmonary infarction three weeks later.

300 Thayer St. (6).

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# Clinical Notes, New Instruments and Techniques

## UREA IN MÉNIÈRE'S DISEASE, MIGRAINE, AND ALLIED CONDITIONS
### Preliminary Report

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Javid and Settlage[1] in 1956 discussed the use and efficacy of urea, intravenously, for the purpose of reducing cerebrospinal fluid pressure, and suggested the possible value of the use of urea in Ménière's disease and various intracranial conditions. In this respect, therefore, I wish to bring attention to a report by Brown,[2] in 1943, who accidentally discovered the curative effect of urea, following a urea-concentration test, in a patient who had suffered severe headaches of a migrainous nature for several years and who was hospitalized for investigation with a view to operation for cerebral compression for relief of his headaches. Upon continuation of 20 grains (1.3 gm.) of urea daily the headaches did not return, and the patient remained free from his migraine for nine years, except during periods when he gave up the regular use of urea. Brown reports three other cases of migraine with marked relief. This author continued to prescribe urea for migraine headaches and for persistent headaches not typically migrainous, with great success. The recommended dose was 20 grain in water three times a day for a week, then 20 grains twice daily for a week, and, subsequently, 20 grains in a single daily dose for an indefinite period. There were no untoward effects. With the administration of intravenous urea, as used in the studies of Javid and Settlage, one would expect more rapid relief in these cases.

These observations have been repeated by Dorothy A. Campbell[3] with considerable success in migraine cases (though larger doses were used than those Brown recommended). She believes that, if given in sufficient quantity over a long period, urea will lessen both the severity and frequency of the attacks. She also agrees to the relatively harmless effect of urea.

In view of these reports and studies, there appears to be a similarity of pathogenesis and biochemical reactions in migraine and Ménière's disease, and therefore it occurs to me that urea should be of equally great value in the relief of Ménière's disease. It has been shown that there is an increase of intralabyrinthine fluid and pressure in Ménière's disease, similar to the cerebral arterial spasm and subsequent edema that occurs in migraine, while in both conditions there is retention of blood sodium and chloride. Thus the treatment of Ménière's disease by the Furstenberg regimen, which consists essentially of a salt-free diet and the ingestion of large amounts of ammonium chloride, is based on these facts. I therefore assume that urea can also be used in Ménière's disease with the expectation of great relief, and dramatic relief, when administered intravenously.

Glaucoma is another condition in which one might expect relief from the (intravenous) application of urea. Reducing serious, increased intraocular fluid pressure in acute congestive glaucoma by intravenous sorbitol solution or 50% sucrose solution is an accepted and valuable treatment. Urea, however, does not cause the renal complications of sucrose. There are other plausible assumptions for the beneficial effect of urea in glaucoma on the basis of the experimental studies of Fremont-Smith and Forbes,[4] who have shown that increased intraocular fluid pressure is associated with increase of intracranial fluid pressure and both are reduced by the injection of urea (50% solution administered intraperitoneally in animals). These authors state, "The foregoing experiments show that, although the intracranial and intraocular pressures are not directly dependent on one another, changes of hydrostatic or of osmotic pressure induced in the blood produce a parallel change in pressure in the eye and in the cranium . . . ." These observations serve to emphasize again the similarity in the mechanisms for formation and for absorption of intraocular and intracranial fluids, and to indicate that these mechanisms are fundamental in the fluid exchange of the body.

In my excursion in serendipity, it also appears plausible, from a standpoint of biochemistry, that urea may also prove of great benefit in the treatment of allergic states,

Accepted for publication April 28, 1956.

since the essential feature of its pathology is edema. This idea becomes more provocative for investigation when one compares the similarities of biochemical reactions in migraine and those of allergic states. In fact, many investigators consider migraine and Ménière's disease allergic manifestations. Possibly the key to much of the mystery of these so-called "allergies" is locked in the relationship of urea to body chemistry to a great degree and to its effect in the regulation and control of edema.

In conclusion, I believe that the use of urea opens up a vast new horizon, never before exploited, in the understanding and control of conditions in which there is edema.

212 W. Wisconsin Ave. (3).

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# Abstracts from Current Literature

## Ear

PRESENT STATUS OF THE OPERATION FOR MOBILIZATION OF STAPES. RICHARD J. BELLUCCIE, Laryngoscope 66:269 (March) 1956.

This is a study of 100 cases at the Manhattan Eye, Ear, and Throat Hospital. The stapes were mobilized in 63 cases, fractured in 35, and found to be already mobile in 2. Of the 63 in which it was mobilized, 35 obtained satisfactory hearing. Six of these, however, returned to preoperative levels in six months, leaving but twenty-nine with continued good hearing. The reasons for this are discussed. However, the oldest cases are but of 18 months' duration.

No consistent preoperative audiometric findings indicated the possibility of success or failure of the operation, and, likewise, there were no very apparent relationships between the duration of the disease or the age of the patient and the success or failure of the operation. If tinnitus was present, it was relieved in about half of the successful cases. No serious complications were encountered. It was found that the mobilization operation did not interfere with a subsequent fenestration. It should be suggested to the patient that both procedures may be necessary for hearing rehabilitation.

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PRESENT STATUS OF STAPEDOLYSIS (STAPES MOBILIZATION). VICTOR GOODHILL, Laryngoscope 66:333 (April) 1956.

One hundred eighty-nine cases are analyzed. Because mobilization restores the normal sound-conduction mechanism, hearing may be raised up to and sometimes a little above the bone-conduction level, while in fenestration the most one can achieve is hearing slightly below the bone level. For this reason and because of the comparative simplicity (to the patient) of the procedure there is a greater flexibility in the number of cases which may be mobilized.

The technique employed for surgical audiometry is described. The "equivalent speech reception threshold" described by Fletcher is used. Four readings are taken during the procedure. It is by these that one decides whether or not enough lysis has been obtained or whether one should try to achieve more.

The operative technique is described; transincudal pressure is first applied, usually in a posterosuperior direction. If this is not successful, a microvibrator is then used. If the incudostapedial joint is a loose one, pressure or vibration is applied directly to the head of the stapes. Sometimes footplate lysis is attempted.

Surgical problems and complications are discussed. These include incudostapedial dislocation, fractures, hemorrhage, vertigo, and perforations. The complications were few and minor.

Results are analyzed. Success is considered in a different light than that for fenestration: It may be not only hearing 30 db. or better but also any closure of the air-bone gap, even though the end-result is below 30 db. Fifty-six per cent of the most recent group (eighty-nine cases) were successful.

Discussion of failures is included as well as illustrative individual case reports. The selection of candidates is still uncertain. No definite criterion is yet available that will enable the operator to predict the chance of success.

HITSCHLER, Philadelphia.

THE PROBLEM OF OTITIS EXTERNA. CHARLES KEOGH and BRIAN RUSSELL, Brit. M. J. 1:1068 (May 12) 1956.

The authors define otitis externa as a dermatitis of the meatus, the concha, or the entire external ear. Five tables are set up to show the contrast between patients (aged 9-79 years) seen by dermatologists and those seen by otologists, and the bacteria found. The only significant difference was the "relatively heavier" infection with cocci and, particularly, with intestinal organisms of the dermatologist's patients. It is said that mycotic causes are not frequent in the British Isles but that fungi are prevalent in hot, humid climates. Three figures in black and white aim to show the various types of involvement of the external ear and canal. Dysfunction of the ceruminous glands is described, and its relation to emotional disturbances is mentioned. "The cause of itching is obscure," but the sudden emptying of ceruminous apocrine

glands in states of fear or pain may be relevant. "Discharges from the middle ear are rarely the cause of otitis externa." This is certainly not the view held in the United States. Climate may be contributory, and dermatoses elsewhere in the body may have a connection; but it is not common to find avitaminosis, anemia, metabolic disorders, or general diseases of the body in correlation. Hair dyes, hair nets, perfumes, and spectacle frames may be causative agents. Prevention depends on maintaining the integrity of the meatal skin, and personal hygiene is all-important. In summary, keep the hands clean, wash after defecation, cut finger nails short and keep the crevices clean, care for any associated skin lesions, avoid scratching the ears, and plug the canals with cotton-wool when bathing, etc.

Treatment calls for prevention, reestablishing the natural protective mechanisms of the skin, destroying the causative agent, controlling itching, and preventing trauma and bacterial invasion. Massaging the canal with olive oil with precision and discretion and applying wool fat (lanolin) or almond oil as a coating "is of the greatest assistance." When there are crusting, bleeding, and swelling, meatal cultures should be taken to determine the nature of the enemy. But indiscriminate use of antibiotics must be discouraged because of sensitization of the skin and eventual ineffective tolerance. Through trial and error one must find the right antibiotic for systemic use; e. g., fecal streptococci may be overcome by streptomycin sulfate, Proteus by chloramphenicol, etc. Transfer by fingers from anus to nasal vestibule to meatus is likely. There is almost positive proof in many instances, for example, where anal pruritus and otitis externa exist simultaneously. The point is made that persons with such infections should not be allowed to handle foods!

ABSTRACTER'S COMMENT: External otitis and meatal conditions are too often called "eczema." Probably few of them are eczematous. The cause is likely to be found by incursive laboratory studies, repeated many times. Few cases are due to general bodily dyscrasias. After the enemy is made known we have many ways of dealing with it. Chemotherapy, iodine, for example, may give astonishing results. Hexylresorcinol can be useful. Scarlet red helps to rebuild skin after exfoliation. X-ray exposures have been useful in some cases but must be used with great discretion. These authors say nothing about irritating soaps getting or being put into ear channels. Mothers who are devoted to soap and water douse them into children's ears. They would not put these into the eyes! This article by the British authors is exhaustive and detailed but adds little to what we already know about "The Problem of Otitis Externa."

I. W. VOORHEES, New York.

THE PRESERVATION AND RESTORATION OF FUNCTION IN OTOLOGY. E. G. COLLINS, Proc. Roy. Soc. Med. 49:63 (Feb.) 1956.

This is Collins' presidential address before the Section of Otology. He refers to the philosophy of Maimonides, who considered truth as relative to present "facts," which may change with years of experience. Taking up first the external ear, the author describes a "chronic hypertrophic eczematous condition of the outer ear" which produces stenosis and partial deafness. He does a kind of atticoantrotomy to restore the canal and enlarge it. The cavity is then lined with a split-thickness skin graft. The results are better in the dry, scaly type rather than in the moist type. "The operation was always carried out under cover of penicillin." Four cases showed resultant improved hearing. In acute otitis media, Collins believes in early paracentesis rather than depending too much on the antibiotics. Permanent damage to the hearing may ensue if incision of the drum and satisfactory drainage are neglected. Chronic adhesive processes are likely to occur in the middle ear after infections. As for the chronic ear, good hearing depends, in general, upon a mobile intact drum, a completely functioning ossicular chain, and an unimpaired space at the round window, with a functioning Eustachian tube. Any single one of these principles, or any combination of them, if violated, may impair the hearing. Suppurative otitis of the chronic type should be dealt with by radical surgery, although it is fashionable to do some modified, conservative operation. True, the latter often works, but it may not succeed as well as if complete exenteration had been done. We are now blessed with excellent magnification, the audiometer, and the antibiotics, all of which help in determining the type of operation, and the ultimate results to hearing. Therefore, we seldom do laborious operations on the labyrinth or petrous apex, as compared with former years. However, a dead labyrinth had best be removed, since meningitis may complicate a seemingly harmless, nonfunctioning labyrinth. Citing Cawthorne, it is related that among 2000 patients there were 47% with otosclerosis. Simson Hall and Cawthorne both believe that otosclerosis is "an abnormality of the mesenchyme" predetermined at birth. But we still do not know the

activating factor. The American, E. P. Fowler Sr., is quoted on the necessity for the earliest possible determination of acuity of hearing in children so that proper management may be at once instituted. The inner ear will always be a problem to the otologist, for obvious reasons. When there is degeneration and atrophy of the organ of Corti, for whatever reason, we are dealing with an irreversible deafness. Our efforts should be directed to prophylaxis and in making useful such elements of hearing as remain. Perceptive deafness, formerly common, has been notably diminished because of reduction of infantile diseases: scarlet fever, measles, and mumps. Inner-ear complications from them are almost rare these days. Industry is responsible for some toxic types of deafness (lead, phosphorus, carbondisulphide in rubber factories, etc.), but these, too, are being well managed. The metabolic disorders, hypothyroidism, hyperthyroidism, and avitaminosis, require skillful diagnosis and management. Noise in industry is being intensively studied with a view to reducing the degeneration of the auditory nerves from percussion. The fatiguing effect of noise is well known. Management of deafness in the young child was mentioned above. We should solicit the aid of public health authorities, teachers, and psychologists in attacking the problem as early as possible. Ménière's disease still baffles us, but some advance in its cure has been made through surgery plus medication. Two theories of causation are held: the neurovascular and the allergic. Collins thinks that the British incline more to the former theory and Americans to the latter. This is certainly disputable, but he seems not to know that S. Rosen has done a fairly minor operation on the tympanic nerves with some relief. This is done instead of the radical labyrinthectomy, which obliterates the vestibular and auditory functions completely. The American, K. M. Day, of Pittsburgh, is mentioned as finding diathermy of little use. Ménière's disease may, of course, be bilateral. Some 37 references to the literature and 10 figures help to illustrate the author's text, which is explicit, self-contained, and admirable in concept and execution.

I. W. VOORHEES, New York.

ETIOPATHOLOGIC BASIS FOR THE TREATMENT OF LABYRINTHINE VERTIGO. R. POCH VINALS, Acta oto-rino-laring. ibero-am. (Barcelona) 7:5, 1956.

Labyrinthine vertigo is considered first from the standpoint of true hydrops of the labyrinth. This is considered a true Ménière's disease. Poch Vinals discusses the pathology and physiology, routine examination (including the electroencephalogram and the relation of the pressure of the central artery of the retina), the various audiograms, and the vestibular tests. He then outlines the treatment, which includes the use of hypertonic saline solution and distilled water, the Migynd and Dederding regimen, Furstenberg's regimen; also the various types of medical treatment, including the use of antihistamines, dimenhydrinate (Dramamine), histamine, nicotinic acid, etc. Finally, he includes the various surgical techniques that have been recommended, with an evaluation of their results.

The second portion of the presentation deals with Ménière's syndrome. This includes neuronitis of the eighth nerve, postural vertigo, and vertigo of radicular origin. Here, again, under these classifications he discusses etiopathology, differential diagnosis, and both medical and surgical indications for treatment. Considerable detail has been placed on the various methods of treatment, with a careful evaluation of the results as reported by the proponents of each specific treatment.

PERSKY, Philadelphia.

DEAFNESS AS A RESULT OF VIRUS INFECTIONS. G. CLAVERIE, Rev. laryng. (Bordeaux) 77:116 (Jan.-Feb.) 1956.

The author discusses the various types of virus infections and their probable relationship to secondary deafness. The various routes of transmission and areas of affection are discussed, but the actual pathologic process in each case is more or less conjectural. He has made the following observations: Postnatal German measles occasionally have been reported to produce deafness; measles rarely produce deafness; chicken pox rarely produces deafness; grippe does occasionally, but it is probably due to the virus per se; epidemic encephalitis produces it rarely; poliomyelitis deafness is rare; herpes zoster deafness is probably due to a herpes of the ganglion of Corti, and in mumps the deafness is probably due to a polioencephalitis. The author feels that the resistance to a viral infection by each nerve is variable and that some may be more susceptible than others. He feels that the facial nerve often shows a greater sensitivity to viral infections than does the auditory nerve.

PERSKY, Philadelphia.

## Larynx

A Study of the Role of Certain Factors in the Development of Speech After Laryngectomy: 2. Site of Pseudo Glottis. Evelyn Y. Robe, Paul Moore, Albert H. Andrews Jr., and Paul H. Holinger, Laryngoscope 66:382 (April) 1956.

It is difficult to determine exactly the method and site of production of voice by the pseudoglottis. The substitute voice may be classified as the buccal voice, the gastric voice, the pharyngeal voice, or the esophageal voice. Each is described. Kallen uses the term alaryngeal speech to include all sounds or whispers made without a larynx. Numerous sites of the alaryngeal speech are mentioned and discussed. Still and motion pictures together with sound tape recordings, as well as x-ray data, were used in the study. Many of the subjects had first to be trained especially. After consideration of all the data available, it was concluded that for the alaryngeal speech to be satisfactory there must be a well-defined controllable constriction zone in the region of the cricopharyngeus, which will act as a pseudoglottis, and an adequate-sized air column below the pseudoglottis.

Hitschler, Philadelphia.

Cancer of the Larynx: Laryngectomy After Radiotherapy. W. A. Mill, Proc. Roy. Soc. Med. 49:73 (Feb.) 1956.

Mr. Mill is president of the Section of Laryngology. He spoke at the meeting held on Nov. 4, 1955. Well over 500 cases of cancer of the larynx have been seen at the Royal Marsden Hospital alone, to say nothing of many other institutions. His own group comprised 47 patients, 3 of whom were women. The youngest patient was 35, the oldest 75! He notes that diagnosis is sometimes "most difficult"; even biopsy may be misleading. Perhaps one should depend more on clinical acumen than on other data. Of course, all evidence which can be acquired must be used. One case seen had tuberculosis of the larynx in symbiosis with cancer. "Glottic growths are by far the most common and . . . the results in these cases are by far the best. In supra-glottic and sub-glottic growths there is much more risk of recurrence." The author is struck by the fact that hoarseness or huskiness is in the history of so many patients, often enduring for years. Negative biopsies may delay operation too long, and, again, clinical judgment had best prevail. At operation one should always examine the carotid sheath for small metastases. These sometimes have yielded to radiotherapy. The author states: "I do not believe in so-called prophylactic block dissection of cervical glands; in the old this operation is very disabling." As for radiotherapy, there may be slow healing in doing surgery after this modality has been used. The operation is more difficult because the skin is hard and fibrous, which is also true of the fascial layers. The infrahyoid muscles may be quite white from fibrosis. General anesthesia is preferred (type is not stated). Low blood pressure technique was tried, but Mill no longer uses this method. In one instance where hexamethonium was used the patient was thought to have died, but was revived; the operation was completed, and all is well after four years. The Sorensen flap is commonly preferred, but variations are allowable. After operation, penicillin into the muscles is given for five days (amount not stated). This drug and sulfathiazole are powdered into the wound. Lateral drains are inserted in the neck during closure of the wound. Blood must be thoroughly removed from the trachea, and sutured edges must not be violated by hemostats, since a good blood supply is paramount. Dead space is avoided by firm bandaging. A polythene tube is inserted through the nose, and all nourishment is by gavage. Breathing exercises are taught and applied before and after operation. The patient is encouraged to get out of bed the day after surgery. Disabilities after operation are cough, involuntary sneeze, flatulence, and tracheitis. Colds, with all their disturbing elements, may vitiate much hard work. The stoma may contract unduly during healing. "Tubes worn to keep the stoma open cause coughing and ulceration and crusting in the trachea with diminution of the lumen."

I. W. Voorhees, New York.

Histopathology and Histogenesis of the Polyps and Singer's Nodules in Comparison to Other Tumors and Pseudotumors of the Larynx. Jean Leroux-Robert, Rev. laryng. (Bordeau), Supp. 60, p. 737, 1955.

The author describes the pathology of the vocal polyps and singer's nodules. It is his observation that these are primarily inflammatory. He further states the following: 1. The terms laryngeal polypi and vocal nodules correspond to a specific clinical entity and histolog-

ically are clearly defined. 2. This classification should be considered entirely separate from angiomas, lipomas, adenomas, myxomas, papillomas, and, in general, all other tumors which may be of a true benign nature. 3. It seems that the laryngeal tumors, and particularly the pseudotumors, are characterized by a local inflammatory process, which may be either exudative or even present a degeneration of the laryngeal submucosa. These do not undergo a spontaneous regression but rather, on the contrary, become organized. 4. Pathologically one can state that the polyps are developed as a consequence of vibratory alterations of one of the vocal cords, which may occur as a result of either a local recurrent irritation or it may be due to some local regional inflammatory process.

PERSKY, Philadelphia.

## Nose

FUNDAMENTALS OF MODERN ALLERGY: SURGICAL TREATMENT OF SINUSITIS ASSOCIATED WITH ASTHMA. RUSSELL CLARK GROVE, New York J. Med. 56:726 (March 1) 1956.

This is one of a series of articles sponsored by the New York Allergy Society, and it is based on 300 patients seen at Roosevelt Hospital. Grove reminds us that every wheeze is not due to asthma. Diagnosis by exclusion will eliminate the presence of a foreign body, tuberculosis, or cancer of the lungs. Then skin and other tests must determine foreign antigens (pollens, dust, etc.) and bacterial allergies. X-rays, with or without radiopaque instillations into the sinuses, may throw much light on these cavities. When surgery is indicated, it must be complete for all areas involved. It is useless to wash out sinuses and remove a polyp or two. "The group which had complete surgery showed twice as much improvement as that with incomplete surgery." Follow-up has gone on for 23 years! Hyperplasia is common, but suppuration is not commonly seen save after an acute infection. Antibiotics and steroid hormones are palliative, while surgery shows improvement in asthma in 69.3% of the total 300 patients!

ABSTRACTER'S COMMENT: Radical surgery of the sinuses for asthma goes back many years. With the coming of the antibiotics and pollen (and similar) tests, there has been a turning away from much more than casual operations. Some improvement has followed, and the asthmatic is always grateful for any relief. The logical course would seem to be to try palliation first, then surgery as indicated, and then back to medical measures if relief is otherwise inadequate.

I. W. VOORHEES, New York.

## Miscellaneous

SENSORY NEUROPATHY ASSOCIATED WITH CARCINOMA OF THE OESOPHAGUS. M. C. H. DODGSON and H. L. HOFFMAN, Ann. Int. Med. 38:130 (Jan.) 1953.

Up to the present time two histologically proved cases of carcinomatous sensory neuropathy have been recorded (Denny-Brown). In both of these, and in the other cases of pure spinal neuropathy, reported by Wyburn-Mason and Lennox and Prichard, the growth was invariably in the bronchus. The case reported here suggests that a similar neuropathy may be associated with carcinoma in other sites.

Dodgson and Hoffman describe a case of sensory neuropathy in a 54-year-old white man occurring in association with carcinoma at the upper end of the esophagus.

The mode of production of sensory neuropathy is still unknown. The available evidence seems to suggest that carcinomatous neuropathy arises as the result of a metabolic disorder, for the production of which a carcinoma is responsible. There is also the possibility that sensory and other neuropathies in association with malignant disease indicate the invasion of the nervous system by a virus.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

BELL'S PALSY TREATED WITH CORTISONE. H. H. ROTHENDLER, Am. J. M. Sc. 225:358 (April) 1953.

Because Bell's palsy may be a result of nerve compression produced by nonspecific inflammatory changes, cortisone was used in the treatment of seven patients with this condition. Treatment was started from 1 to 10 days after the onset of the paralysis and was continued for 10 to

14 days. In all but one case improvement started in 1 to 5 days and complete recovery occurred in from 7 to 12 days after cortisone treatment was begun.

BERLIN, Mount Vernon, N. Y. [A. M. A. ARCH. NEUROL. & PSYCHIAT.].

A SUPERIOR NEW NASAL DECONGESTANT: CLINICAL EVALUATION OF TYZINE IN SIX HUNDRED SEVENTY-FIVE PATIENTS. HAROLD C. MENGER, New York J. Med. 56:1279 (April 15) 1956.

Menger previously reported on the use of Tyzine in 203 cases with "nasal congestion." This is a new sympathomimetic amine found to be exceptionally effective and well tolerated when used topically in the nose. The present article extends the number of patients treated to 675, of whom 90 were children. The drug is prepared in the laboratories of the Chas. Pfizer & Company, Inc., Brooklyn. It is a synthetic with a long chemical name. The usual duration of treatment, by drops or sprays, was from 4 to 10 days (610 patients) and from 1 to 3 days or to 10 days, according to the need. Some 450 adults with hypertension were observed at intervals of 10 minutes to several days. Excellent relief was afforded in 95% and fair relief in 3%. Relief for four hours was often obtained. No adverse effects were noted save some drowsiness in children, which promptly disappeared when the drug was stopped.

ABSTRACTER'S COMMENT: Physicians' offices are being fairly flooded these days with decongestants, from drops to spray bottles. All of them open the nose, and are helpful preceding office treatment, irrigations, etc. People who have nasal obstruction will "do anything" to get relief. Few of these constricting agents have any antiseptic value, although one, at least, has the word "antibiotic" on its label. Little is said concerning etiology, which is still basic in all medicine. In years past, anesthetization and cauterization of the inferior turbinates brought more than temporary relief. It is still a good procedure.

I. W. VOORHEES, New York.

PNEUMOTHORAX FOLLOWING OPERATIONS ON THE LOWER NECK. FRANK C. MARCHETTA, New York J. Med. 56:1287 (April 15) 1956.

Pneumothorax has occurred four times during the last 150 radical surgical procedures in the Head and Neck Service of the Roswell Park Memorial Institute, Buffalo. The four are reported in detail, with x-ray reproductions of the chest pathology. Pediatric literature contains many reports of pneumothorax following tracheotomy. Children under 4 years are likely to be victims. Barrie, Keis, and others have reported deaths from tension pneumothorax during low neck surgery, especially after thyroidectomy. Four paths of ingress are described: 1. Air may enter directly through the cupola of a lung. 2. During bronchoscopy the mucous membrane may be ruptured, giving an open route to the submucosa. 3. Air may enter the interstitial tissues of a lung from ruptured alveoli and dissect along, making deep pathways as it proceeds. 4. It may enter through a surgical wound and enter the superior mediastinum. Loose tissues may be seen to retract during inspiration, and a sucking noise may be heard. Pneumomediastinum may well precede pneumothorax, with rapid and progressive worsening of the patient's condition, and death. Aspiration by large-bore needle in the chest wall or catheter introduction may be lifesaving; the catheter is preferred, since needle aspiration may be insufficient, and recurrence of air may be lethal. House physicians should be on the lookout for pneumothorax and not wait for the attending surgeon to come in. A matter of minutes may spell fatality.

I. W. VOORHEES, New York.

SPECIFIC INJECTION TREATMENT OF HAY FEVER AND ASTHMA. SELIAN HEBALD, New York J. Med. 56:1299 (April 15) 1956.

This is another in the series on modern allergy, prepared under the editorship of Dr. Samuel J. Prigal. Hebald is affiliated with the Roosevelt and Joint Diseases Hospitals. Aside from prevention, where external causes can be eliminated, the best available means of protection against hay fever and asthma are specific injections. Pollens, house dust, feathers, and danders are the basic elements plus the spores of molds. Occupations such as upholstering are poisonous to many workers, particularly those who inhale dust from kapok, raw cotton, etc. As for cereals, those who cannot tolerate powders from them can ingest these foods with impunity! The main principle of treatment is to inject small doses subcutaneously, increasing such to the point of intolerance, then desisting for a time, repeating the series as

necessary. Table I in the article gives the average dosage according to sensitivity. Injections are given at 4- to 7-day intervals. One should give a minimum first dose and then proceed cautiously to the possible tolerated maximum. The patient must report his reactions as instructed by the physician. Children usually take the dosages well. About one-third of all hay-fever patients have asthmatic symptoms some time in their lives. Fortunately, relief can be obtained in 85%. When hay fever is satisfactorily treated, colds and sinusitis are less frequent. Bacterial allergies require a somewhat different approach. Autogenous vaccines are to be preferred, but good results may be had with stock supplies. These autogenous preparations may be very potent and should be administered with caution, diluted down to 1:1000 from the 2 to 5 billion killed organisms, as the preparation comes from the laboratory. In general, all reactions can be observed in from 1 to 30 minutes, and so it is well to have the patient wait so that the loci can be "read." Itching, sneezing, and coughing may be somewhat disturbing, but they soon pass off as a rule. In severe reactions one should apply a tourniquet proximal to the body, assuming that one is using an arm for injections. This is done to slow down the absorption. Then one should inject about 0.4 ml. of epinephrine, 1:1000, above the tourniquet or in the opposite arm, releasing the tourniquet gradually. At the succeeding visit it is well to reduce the dosage to a quarter of the disturbing amount. Very hot weather seems to increase the severity of reactions, possibly because of peripheral vasodilatation. We can often desensitize the patient to a point where he is grateful for relief, but he is seldom "cured" in the sense that no recurrence is likely. It is well to explain this fact to the too-optimistic patient.

ABSTRACTER'S COMMENT: Treatment of allergy calls for no end of patience on the part of doctor and sufferer. Even the correct diagnosis may consume much time and effort. Then, the responses may be too great or too small, so that trial and error become the working principles, thus exacting sharp judgment and a refusal to be discouraged. Instead of the subcutaneous method of injections, some authorities prefer the intradermal technique because the antigens seem to be more readily produced and the dosage need not be so large. This is, of course, a matter of opinion based on individual experience.

I. W. VOORHEES, New York.

# Books

**Lungenkrebs und Bronchographie (Cancer of the Lung and the Bronchogram).** By H. Anacker. Price, DM 28.50 ($6.80). Pp. 78, with 45 illustrations. Georg Thieme Verlag, Herdweg 63, (14a) Stuttgart, 1955.

The author presents an excellent monogram in which he discusses the problem of bronchogenic carcinoma. He evaluates its development and progress. He describes the technique of bronchography and stresses four important diagnostic measures: the bronchogram, the tomograph, bronchoscopic examination, and, finally, the cytologic examination. He also includes a great number of sketches indicating the possible origin of bronchogenic carcinoma. These, together with photographs of both the bronchograms and the pathologic preparations, help clarify the value and the importance of his technique. He also discusses, among other influences in the development of carcinoma, the role of cigarette smoking. This is an excellent monogram, valuable to the roentgenologist, bronchoscopist, the internist, and the chest surgeon.

**The Surgical Treatment of Otospongiosis.** By Maurice Sourdille. Price, not given. Pp. 253, with 52 illustrations. Masson & Cie, 120 boulevard Saint-Germain, Paris, 6e, 1948.

In this monogram the author has presented a detailed description of his fenestration operation. He describes in detail his early procedures in the two-stage operation, the indications, and results. This was originally presented in 1935. The second section was presented in 1939 as a supplement to his report of 1935. Here, too, he describes in great detail the indications for operation and the modifications in his technique. The third section of this monogram deals with a second supplement to the 1935 report. In view of the fact that the war interrupted much of his research and operative activities, the author states that after the termination of the war he was greatly influenced by the work of American surgeons (Lempert, Shambaugh Jr.). He discusses in this supplement (1) the psychoacoustic problem, (2) the surgical problem, and, finally, (3) the pathology. He also presents some further modifications in his technique and indicates that perhaps the one-stage operation is of greater advantage than his original two-stage operation.

In view of the fact that this has been published in 1948 and that there has been a great modification in our present-day concepts of the pathology, indications for operations, and surgical technique, with a different concept of the entire question of otosclerosis, this monogram, though valuable from the historical point of view, is somewhat out of date.

# Directory of Otolaryngological Societies *

## FOREIGN AND INTERNATIONAL

### Canadian Otolaryngological Society

(Société Canadienne d'Otolaryngologie)

President: Dr. D. S. Gorrell, 110 Medical Arts Building, Calgary, Alta.
Secretary: Dr. G. Arnold Henry, 328 Medical Arts Building, Toronto.

### International Bronchoesophagological Society

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Fourth International Congress of Bronchoesophagology.
President of Congress: Dr. Antonio Carrascosa.

### International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

### International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

### Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Juan Manuel Tato, Azcuenaga 235, Buenos Aires, Argentina.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

### Sixth International Congress of Otolaryngology

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.
Place: Hotel Statler, Washington, D. C. Time: May 5-10, 1957.

## NATIONAL

### American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. John R. Lindsay, Chicago.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.

### American Academy of Ophthalmology and Otolaryngology

President: Dr. Algernon B. Reese, 73 E. 71st St., New York 21.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave., Bldg., Rochester, Minn.

### American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

### American Broncho-Esophagological Association

President: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958 (afternoons only).

### American Laryngological Association

President: Dr. LeRoy A. Schall, 243 Charles St., Boston 14.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Statler Hotel, Washington, D. C. Time: May 3, 1957.

### American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Percy E. Ireland, 170 St. George St., Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

**SECTIONS:**

Eastern.—Chairman: Dr. John A. Murtagh, Hanover, N. H.
Southern.—Chairman: Dr. J. M. Robison, Houston, Texas.
Middle.—Chairman: Dr. William H. Craddock, Cincinnati.
Western.—Chairman: Dr. David D. DeWeese, Portland, Ore.

AMERICAN OTOLOGICAL SOCIETY, INC.

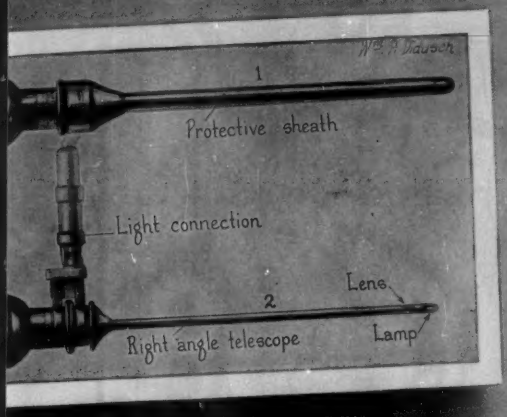
President: Dr. John R. Lindsay, 750 E. 59th St., Chicago 37.
Secretary-Treasurer: Dr. Lawrence R. Boies, University of Minnesota Hospitals, Minneapolis 14.
Place: Statler Hotel, Washington, D. C. Time: May 4, 1957.

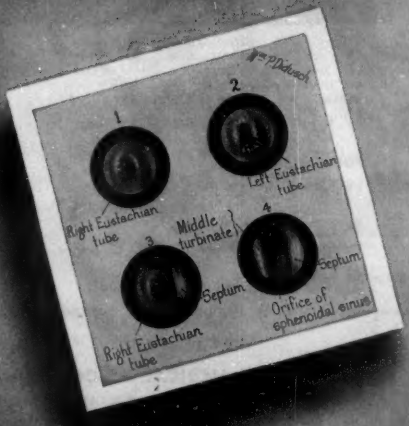
AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Joseph Gilbert.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. Dean McAllister Lierle, University Hospital, Iowa City.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago.



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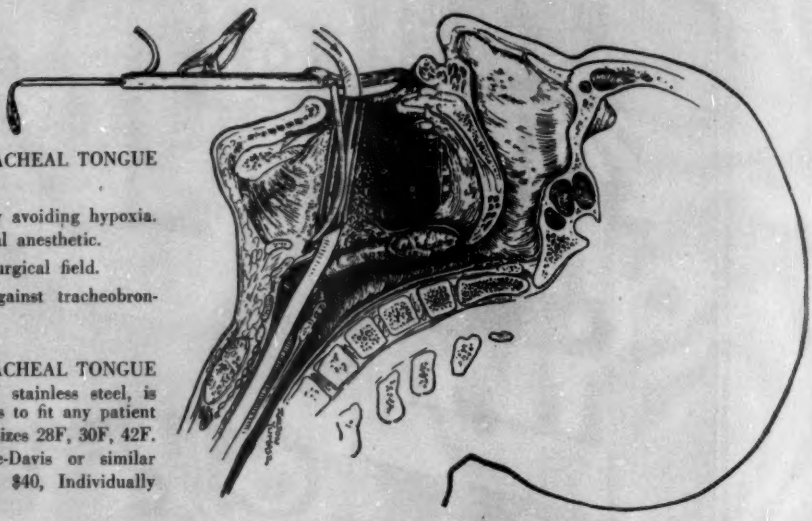

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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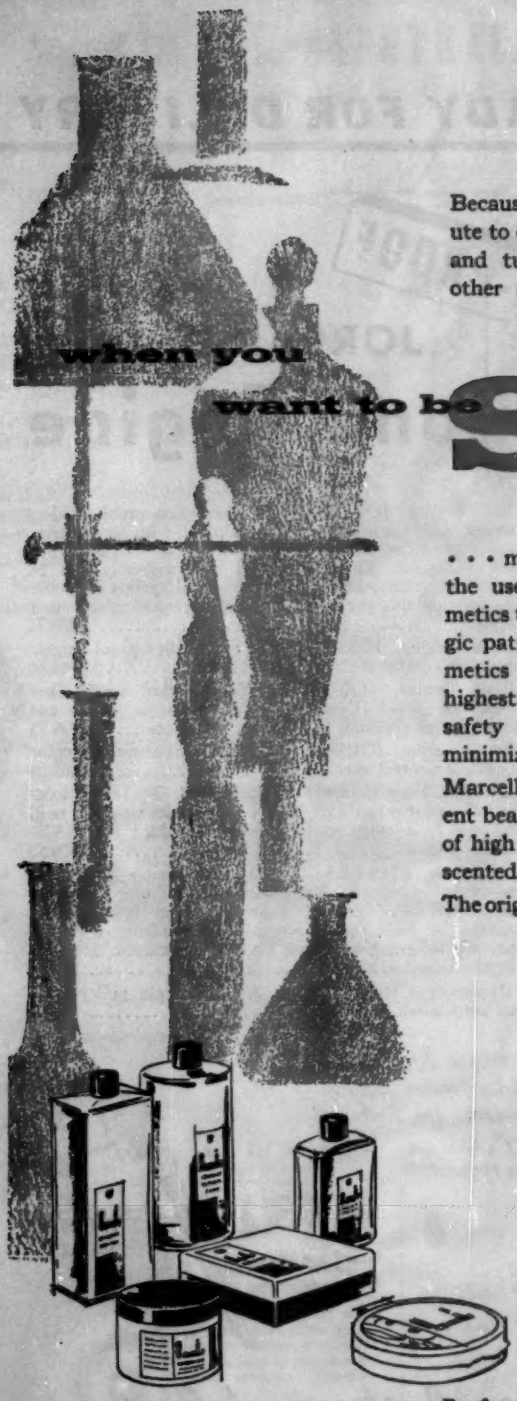

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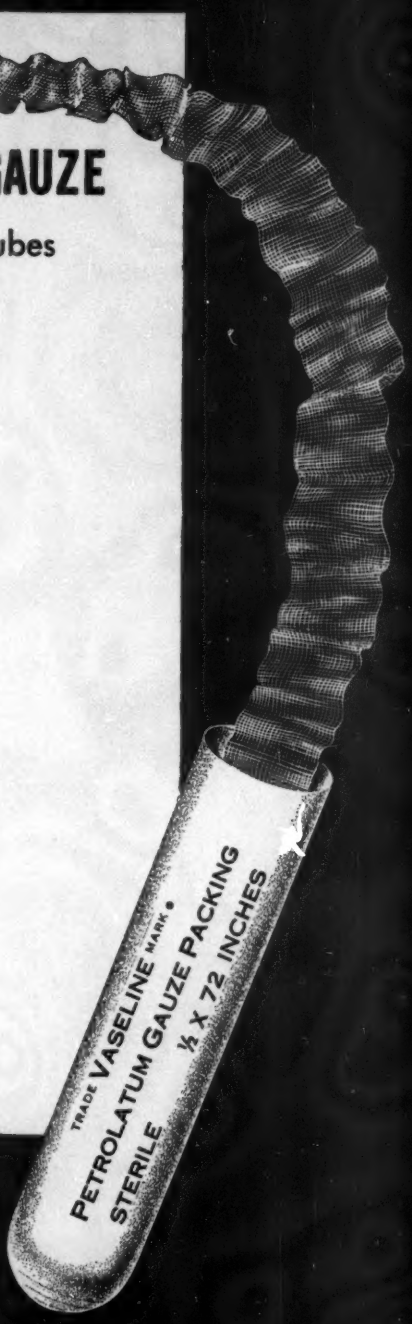


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